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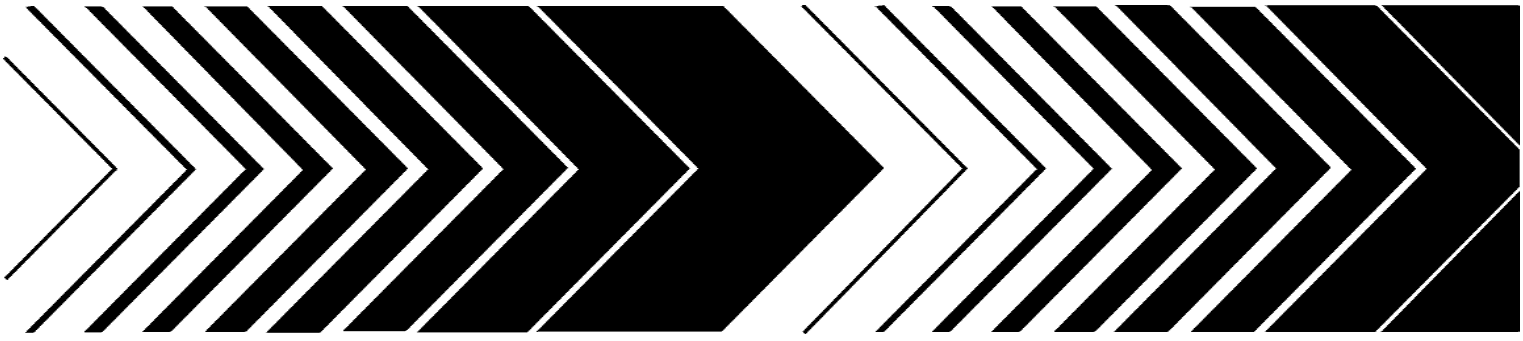
External Review Draft

EPA

Air Toxics Research Strategy

Notice

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.



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Authors, Contributors, and Reviewers

Principal Authors

Dr. William Boyes–National Health and Environmental Effects Research Laboratory (MD-74B)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. John Chang–National Risk Management Research Laboratory (MD-54)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Fred Dimmick–Office of Air Quality, Planning and Standards (MD-14), co-leader,
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Stan Durkee–Office of Science Policy (8104R)
U.S. Environmental Protection Agency, Washington, DC 20460

Mr. Chuck Mann–National Risk Management Research Laboratory (MD-63)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Dennis Pagano–Office of Air Quality, Planning and Standards (MD-13)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Jeffrey Ross–National Health and Environmental Effects Laboratory (MD-68)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Bill Russo–National Health and Environmental Effects Laboratory (MD-51A)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Mary Jane Selgrade–National Health and Environmental Effects Research Laboratory
(MD-92), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Linda Sheldon–National Exposure Research Laboratory (MD-77)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Chon Shoaf–National Center for Environmental Assessment (MD-52), co-leader,
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Daniel Vallero–National Exposure Research Laboratory (MD-76)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Tim Watkins–Office of Air Quality, Planning and Standards (MD-13)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Authors, Contributors, and Reviewers
(cont'd)

Contributors

Dr. Gary Foureman–National Center for Environmental Assessment (MD-52)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Brian Gullett–National Risk Management Research Laboratory (MD-65)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. William McDonnell–National Health and Environmental Effects Laboratory (MD-58B)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Douglas McKinney–National Risk Management Research Laboratory (MD-60)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. James McKinney–National Health and Environmental Effects Laboratory (MD-74)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Mr. Johnnie Pearson–National Exposure Research Laboratory (MD-75)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. John Vandenberg–National Health and Environmental Effects Research Laboratory (MD-51A), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Michael Waters–National Health and Environmental Effects Research Laboratory (MD-51A)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Reviewers

Mr. Bob Hetes–Office of Air Quality, Planning and Standards (MD-13)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Deidre Murphy–Office of Air Quality, Planning and Standards (MD-13)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Roy Smith–Office of Air Quality, Planning and Standards (MD-13)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Michel Stevens–National Center for Environmental Assessment (MD-52)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Authors, Contributors, and Reviewers
(cont'd)

ORD Science Council

Dr. Judith Graham–National Exposure Research Laboratory (MD-75)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Hugh McKinnon–National Risk Management Research Laboratory (MC-235)
U.S. Environmental Protection Agency, Cincinnati, OH 45268

Dr. Robert Menzer–National Center for Environmental Research (8701-R)
U.S. Environmental Protection Agency, Washington, DC 20004

Dr. Jay Messer–National Exposure Research Laboratory (MD-75)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Lee Mulkey–National Risk Management Research Laboratory (MC-225)
U.S. Environmental Protection Agency, Cincinnati, OH 45268

Dr. Michael Slimak–National Center for Environmental Assessment (8601-D)
U.S. Environmental Protection Agency, Washington, DC 20460

Dr. Kevin Teichman–Office of Science Policy (8104-R)
U.S. Environmental Protection Agency, Washington, DC 20004

Dr. Gilman Veith–National Health and Environmental Effects Research Laboratory (MD-87)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

Dr. Vanessa Vu–National Center for Environmental Assessment (8601-D)
U.S. Environmental Protection Agency, Washington, DC 20460

Dr. Harold Zenick–National Health and Environmental Effects Research Laboratory (MD-87)
U.S. Environmental Protection Agency, Research Triangle Park, NC 27711

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EXECUTIVE SUMMARY

The EPA's Office of Research and Development (ORD) has developed a draft Air Toxics Research Strategy (ATRS). This strategy was developed to assist in improving our understanding of air toxics and to manage effectively and efficiently our resources for air toxics research.

National Air Toxics Assessments

The ATRS is needed to address the uncertainty in EPA's National Air Toxics Assessments (NATA). As explained in Chapter 1, these assessments will be used in a number of ways. They will be used in characterizing the residual risks remaining after implementation of the technology-based standards for sources of air toxics. The assessments also will be used to better understand the risks in urban areas, including risks from mobile sources, and the risks associated with indoor air toxics. They also will be used in characterizing the overall air toxics problem and measuring progress towards EPA's program goals, through national-scale assessments. Currently, EPA's initial national-scale assessment under NATA is beginning peer review.

Structure of the ATRS

As shown in Figure 1 and explained in Chapter 2, the ATRS includes input from the air toxics program, in particular, risk estimates and the accompanying uncertainty in these estimates developed during NATA activities. This ensures that the ATRS is relevant to EPA's air toxics program and that the ATRS remains current to the changing facets of air toxics assessments. The research coming out of the ATRS will produce information and tools to reduce the uncertainty in risk assessments for air toxics and to help manage these risks.

The ATRS is based on the risk assessment/risk management framework, key research questions, and strategic principles shown in Figure 2. In designing the ATRS, a risk assessment and management framework was used to organize key research questions and associated research needs. This framework presents a logical approach for assessing and managing risks and provides a basis to ensure that a reasonably comprehensive list of relevant research is identified for consideration.

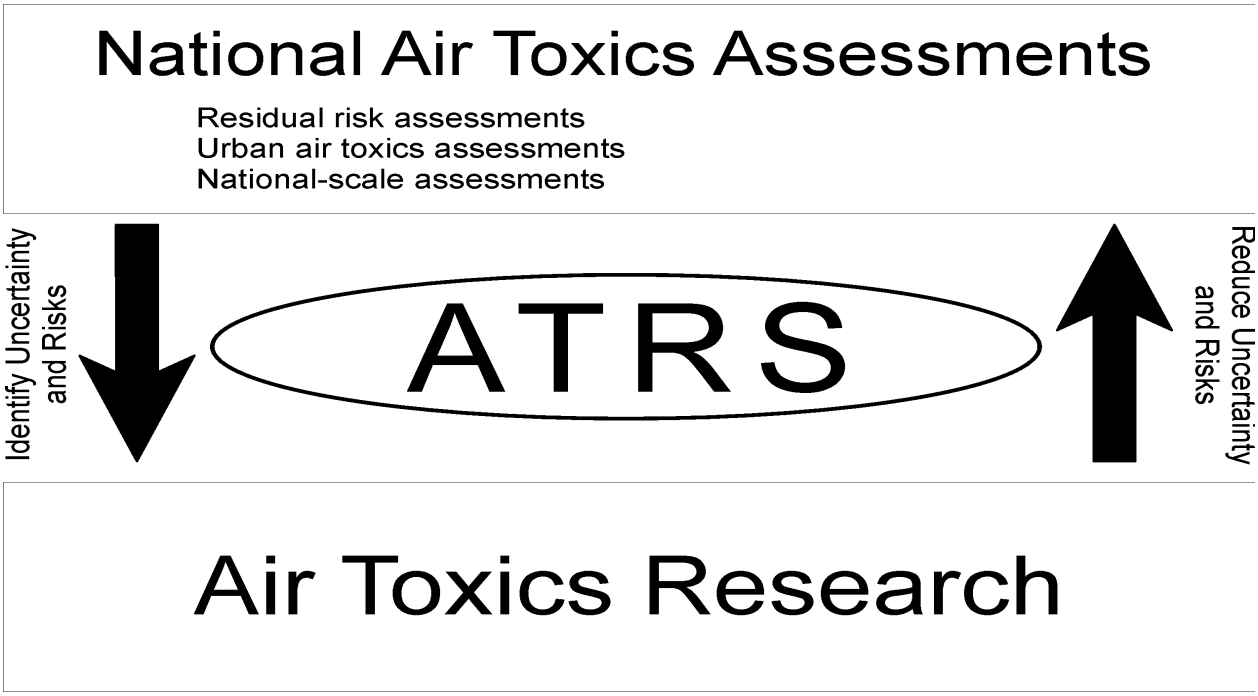

Figure 1. Relationship of air toxics research and NATA.

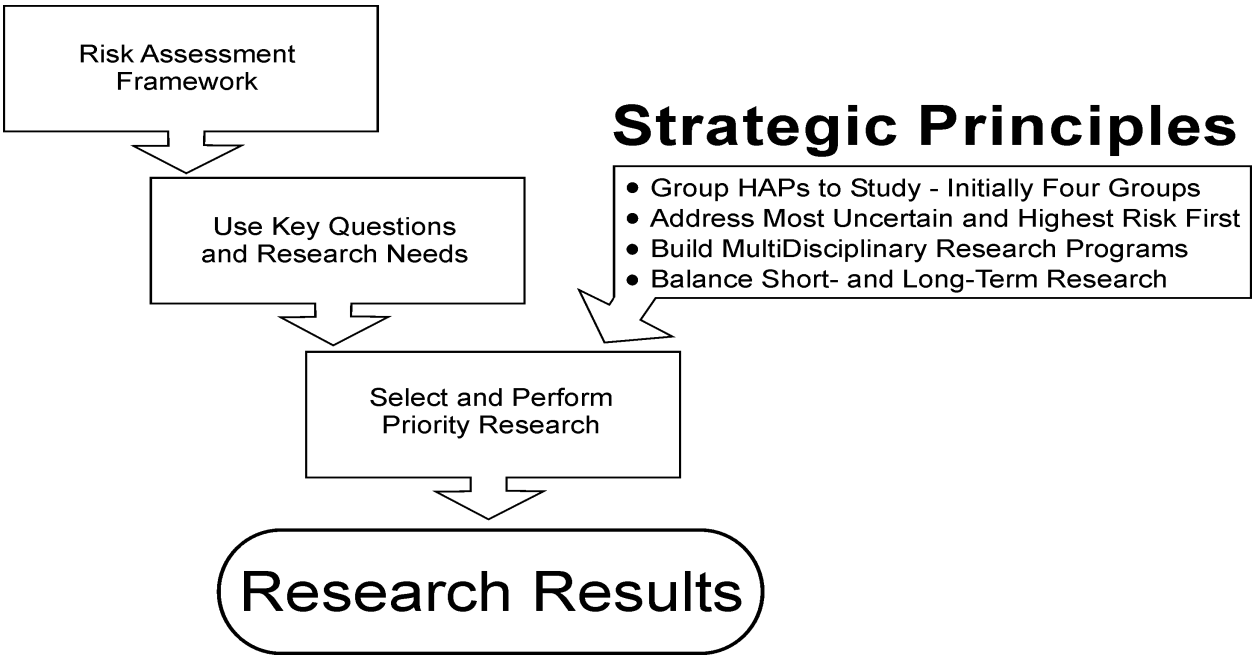

Figure 2. ATRS structure.

Key research questions were developed considering the current scientific knowledge of air toxics and the air toxics program's goals and needs. Key questions are overarching questions and are intended as a departure point for developing and organizing more specific research needs.

Research needs were identified to help answer the key questions. The research needs were selected to be as comprehensive as practically possible, with special emphasis on filling research gaps, improving our understanding of air toxics for each part of the paradigm, and addressing program goals and needs. The research needs are listed in Chapter 2.

Strategic principles were developed to aid in selecting specific air toxics on which to focus initially and in identifying priority research activities. These principles are to be invoked in the selection and prioritization of air toxics research. The principles are listed in Table 1.

TABLE 1. ATRS PRINCIPLES

Principle 1.	Increase the usefulness of the research program by grouping air toxics initially based on physicochemical properties to assist in future studies of structure-activity relationships (SARs).
Principle 2.	Focus research and development on the greatest risks to people and the environment.
Principle 3.	Focus research on reducing major uncertainties in risk assessment and improving cost effectiveness in risk prevention and management.
Principle 4.	Undertake and foster multidisciplinary research.
Principle 5.	Ensuring an appropriate balance between near-term research and long-term research.

The first principle is needed to address the large number of individual air toxics. It is impossible to scientifically evaluate individual air toxics in a reasonable amount of time with foreseeable resources. Grouping of compounds within well established chemical classification schemes used in organic chemistry can have long-term and multiple payoffs in terms of linking research to well established chemical literature on air toxics physical and chemical properties and reactivities that can underlie their biological/nonbiological activities. The next two principles work together to direct research towards air toxics that pose the greatest risk and the

most uncertainty first. The last two principles ensure that air toxics research is completed in a collaborative manner, while avoiding only performing near-term research. All these principles assisted in developing the ATRS and will be used in implementing the ATRS. They were used in selecting four groups of air toxics to study initially.

The Four Air Toxics Groups: Aldehydes, Halides, Metals, and POM/Hydrocarbons

In selecting the initial four air toxics groups using *Principle 1*, factors relevant to EPA's air toxics program and the state of knowledge on air toxics were considered. Appendix B is a compilation of the information used in selecting the four HAP groups. The CAA HAPs and other program relevant air toxics were grouped by SAR, and then program assessments were used to identify the relative significance and uncertainty associated with assessments for air toxics within the SAR-based groups. In addition, EPA's current understanding concerning exposure potential and health risk information for these air toxics was added to the Appendix B table. Although no precise formula was used, consideration of an overall view of the factors (*Principles 2* and *3*) resulted in the selection of four air toxics groups: (1) aldehydes, (2) halides, (3) metals, and (4) POM/hydrocarbons. An example of how the ATRS would play out for POM is presented in Chapter 3. As the ATRS is implemented, EPA will undertake and foster multidisciplinary research (*Principle 4*) and will ensure an appropriate balance between near-term research and long-term research (*Principle 5*) in studying these air toxics groups.

Implementation of the ATRS

As explained in Chapter 4, the ORD expects significant accomplishments as a result of implementing the ATRS. The most significant accomplishments involve reducing uncertainties in the NATA. In addition, ORD will obtain data and identify where the grouped air toxics facilitate an increased understanding of other air toxics within the groups. To ensure a consistent emphasis on air toxics research, a steering committee will be formed to direct detailed planning activities for air toxics and assist EPA research planning and budget development. Scientist-to-scientist meetings will be help to engage principal investigators and other scientists in the air toxics issue and to foster cross-laboratory projects by increasing face-to-face contact between investigators in different labs. These meeting also will bring scientists' expertise in designing and executing air toxics research and will insure that this input is included in implementing the strategy and developing research plans.

1. INTRODUCTION AND RATIONALE

The U.S. Environmental Protection Agency's (EPA's) Office of Research and Development (ORD) is committed to conducting research with the purpose of providing the best science possible to support EPA activities and regulations. The Office of Air and Radiation (OAR) is charged with developing and implementing programs and regulations required under the Clean Air Act (CAA). This document is an ORD research strategy for identifying and addressing air toxics research needs, which will allow ORD to support OAR's activities under the CAA. This chapter describes OAR's plans to implement EPA's air toxics program and presents a framework for identifying the research, in broad terms, needed to support this program. Specific research needs for ORD to address are identified in subsequent chapters of this strategy.

1.1 WHAT ARE "AIR TOXICS", AND WHAT RISKS DO THEY POSE?

Air toxics are air pollutants that may pose a risk to human health or the environment. Air toxics, include 188 chemicals that are listed under Section 112(b) of the CAA as hazardous air pollutants (HAPs) and are, therefore, subject to regulations for stationary sources under the Act. In addition, as part of a rulemaking published in December 2000, EPA listed 21 pollutants as mobile source air toxics. Twenty of these pollutants are among the 188 HAPs listed in Section 112(b) of the CAA. The twenty-first pollutant is diesel particulate matter and diesel emission organic gases (both are considered one pollutant). Additional chemicals and chemical mixtures that are dispersed through the air and present risks to humans or the environment also may be considered air toxics. The EPA estimates that about 4.6 million tons of HAPs were emitted by U.S. anthropogenic sources into the ambient air in 1996. The 1996 base year estimate includes major, area, and mobile sources. (1996 NTI, www.epa.gov/AIRSData/, U.S. EPA, January 2001)

Of the 188 air toxics listed as HAPs, 17 represent chemical groups (e.g., polycyclic organic matter [POM]) rather than individual chemicals. Air toxics include pollutants like benzene found in gasoline, perchloroethylene emitted from dry cleaners, methylene chloride used as an

industrial solvent, heavy metals such as mercury and lead, polychlorinated biphenyls (PCBs), dioxins, and some pesticides. Air toxics include a wide variety of organic and inorganic substances released from industrial operations (both large and small); fossil fuel combustion, including gasoline and diesel-powered vehicles; the use of consumer and commercial products; off-gassing of materials used in buildings; human activities (e.g., cooking and smoking), and many other sources. Air toxics that originate from outdoor sources may disperse locally, regionally, nationally, or globally and, after deposition, may persist in the environment or bioaccumulate in the food chain, depending on their characteristics (e.g., vapor pressures, atmospheric transformation rates, lipophilicity). Air toxics originating from sources indoors may disperse and be transformed within buildings. In addition, air toxics originating outdoors infiltrate to indoor environments, and those from sources indoors find their way outdoors where they may impact the outdoor environment. Humans become exposed to air toxics by coming into contact with environmental media (e.g., air, water, soil) that are contaminated by air toxics. The route (inhalation, ingestion, or dermal absorption) and media of exposure (air, water, food, soil, or dust) will depend on a number of factors, including the physical and chemical properties of the air toxics.

Although air toxics have been regulated as individual chemicals, they usually are released as mixtures. The composition of the mixture will depend on the sources. The understanding of air toxic mixtures becomes important when characterizing risk because health impacts will depend not only on the risk associated with aggregate exposures (one chemical, multiple pathways), but also, on the risk resulting from cumulative exposures (multiple chemicals and multiple pathways).

Air toxics and mixtures containing them have the potential to pose a variety of health risks depending on their toxicity, as well as the magnitude, frequency, duration, and route of exposure. Although EPA has classified more than half of the air toxics to be known or suspected human carcinogens, many also are known to have respiratory, neurological, immune, or reproductive system effects. Health effects are more likely to be seen in more susceptible, sensitive, or highly-exposed populations, such as children, the elderly, and those individuals living in community “hot spots”. The specific human health effects associated with environmental exposures to various air toxics or air toxic mixtures may differ, depending on the particular circumstances of exposure (e.g., the amount of chemical, the length of time a person is exposed, the stage in life of the person exposed). Additionally, many air toxics are known to cause

adverse effects in many fish and animal species (e.g., toxicity in fish, reproductive decline in bird species), including endangered species. These environmental effects may be felt by individual species within a single level of the food chain or by the entire ecosystem, where multiple species are affected.

1.2 EPA'S TRANSITION TO A RISK-BASED AIR TOXICS PROGRAM

With the passage of the CAA Amendments of 1990, a shift in the approach to dealing with air toxics occurred. The current approach to reducing air toxics under the CAA includes the identification and listing of air toxics as HAPs, the development of technology-based standards to reduce HAP emissions, and the implementation of a risk-based program to assess the risks remaining after the implementation of the technology-based standards and to establish additional standards if necessary to protect public health and the environment.

With the July 19, 1999, publication of the *National Air Toxics Program: Integrated Urban Strategy* (Federal Register, 1999; hereinafter referred to as the *Integrated Urban Strategy*), the EPA laid out the framework for EPA's risk-based air toxics program (see Appendix A for additional details). This program is designed to characterize, prioritize, and equitably address exposures to air toxics and their serious impact on the public health and the environment through a strategic combination of regulatory approaches, voluntary partnerships, ongoing research and assessments, and education and outreach. The program addresses air toxic emissions from large and small stationary sources, mobile sources and indoor air sources as part of its strategy for reducing risks from exposure to air toxics. The program also considers the transport and deposition of air toxics to many of the nation's water bodies. The goals set forth in the *Integrated Urban Strategy* are to (1) attain a 75% reduction in incidence of cancer attributable to exposure to HAPs emitted by stationary sources in all urban areas nationwide, (2) attain a substantial reduction in public health risks posed by HAP emission from area sources in all urban areas nationwide, and (3) address disproportionate impacts of air toxics hazards across urban areas.

In addition to the goals in the *Integrated Urban Strategy*, EPA has established goals (see Figure 3) for the air toxics program under the Government Performance and Results Act (GPRA). The initial GPRA goal is based on achieving a percent reduction in air toxic emissions. Because knowledge and tools to assess the impacts of these emissions on public health and the

Initial Emissions Reduction Based Goal

By 2010, Reduce air toxics emissions by 75% from 1993 levels.

⇓

Risk-Based Goal (effective in FY 02)

By 2020, eliminate unacceptable risks of cancer and other significant health problems from air toxics emissions for at least 95% of the population, with particular attention to children and other sensitive subpopulations; and substantially reduce or eliminate adverse effects on the natural environment.

Figure 3. Changing GPRA goal reflects the transition to a risk-based program.

environment currently are limited, this goal reflects the straightforward intent to reduce total air toxics emissions as a means to reduce risks associated with exposure to air toxics. However, as EPA extends knowledge, develops better assessment tools, and begins to address the risks associated with exposure to these emissions as required by the CAA, a GPRA goal directed specifically at risk reductions associated with exposure to air toxics is more appropriate. This risk-based GPRA goal likely will be adopted in 2002 and will guide the program into the year 2020.

1.3 HOW WILL ORD'S AIR TOXICS RESEARCH SUPPORT THE RISK-BASED PROGRAM?

If the EPA is to be successful in implementing its risk-based air toxics program, a critical need will be the continued improvement of the science supporting the EPA risk assessments and risk management decisions. Information on air toxics is incomplete. Although some data exist to evaluate exposure and health impacts of specific air toxics (e.g., benzene), much more information still is needed to conduct reliable quantitative risk assessments for most chemicals (e.g., specific metal species). Even more information is needed to conduct cumulative risk assessments for mixtures of air toxics aggregated over multiple sources and multiple routes of exposure and to understand risks to susceptible populations. This need for additional information is seen across all aspects of the risk paradigm. The research needs identified in this strategy are intended to fill the gaps in current knowledge about air toxics by providing better data (across the source-exposure-dose-response continuum) for use with existing methods and by providing newer methods that will help in the evaluation of toxicity, exposure, and risk. Along

with improvements in risk assessment and characterization tools, improved risk prevention and intervention tools will decrease the uncertainty underlying risk management decisions. This strategy presents the road map for improving the science supporting the EPA's risk assessment and risk management activities.

Most of the EPA's air toxic risk assessments will be conducted by OAR as part of the EPA's National Air Toxics Assessment (NATA) activities. The NATA activities include expanding air toxics ambient (indoors and outdoors) and deposition monitoring; improving knowledge of indoor and outdoor emission factors; improving and periodically updating emission inventories; improving national- and local-scale air quality, multimedia, and exposure models (including models that consider stationary and mobile sources); conducting indoor air quality studies; and improving and using exposure and assessment tools. The results of NATA activities will be used to characterize risks, prioritize risk management efforts, and track progress toward meeting air toxics program goals and objectives. In addition, the NATA activities also may serve to highlight specific research needs by identifying major areas of uncertainties, especially those that may affect policy decisions. Eventually, as the science improves through implementation of this strategy, the assessments conducted as part of NATA will be able to better access multimedia, multi-pathway exposures and risks for chemical mixtures, as well as single chemicals, and will address susceptible populations. ORD research will play a critical role in improving the science and, thus, in reducing the uncertainty in the NATA activities.

The NATA activities will include assessments conducted on a national scale. These national-scale assessments will be conducted at 3-year intervals and will be used by OAR to inform risk reduction policies and to measure progress toward specific risk reduction goals. Research supporting these national-scale assessments under NATA will be continuous. However, at critical points in time, OAR will need to assemble the available information and tools to conduct each national-scale assessment. These critical points in time occur approximately 1 year in advance of each assessment. Currently, national-scale assessments are planned for 2003, 2006, 2009, and 2012 (and every 3 years thereafter), so research input to support these assessments would be needed by the end of 2002, 2005, 2008, and 2011, respectively.

The NATA activities also include various regional- and local-scale assessments. These assessments may be conducted as part of an urban- and community-based pilot or, perhaps, to

provide a more refined assessment of a potential problem that was identified in one of the national-scale assessments. Regional- and local-scale assessments will include consideration of indoor air sources and mobile sources air pollutants deposited to soil and water. These assessments have already been initiated and will be ongoing over the next several years. OAR will use the most current information and tools available at the time to conduct these assessments. States, communities, and Tribal authorities also need information and tools as they conduct their own assessments.

NATA activities also include source-specific risk assessments to determine the risk remaining (i.e., residual risk) after a MACT is promulgated. If EPA determines that the MACT standard does not protect the public health with an ample margin of safety or does not prevent an adverse environmental effect, a residual risk standard is required within 8 or 9 years of promulgation of the MACT standard, depending on when the MACT standard was required. When the technology-based air toxics program is complete, about 100 MACT standards will have been promulgated. The first MACT standard was promulgated in late 1993. Promulgation of MACT standards is expected to continue through at least 2002. Therefore, OAR now is conducting and will continue to conduct risk assessments through at least 2010 to assess the need for additional standards, and, if a need is found, the appropriate control level. Research to support these activities will be needed throughout this time period.

The EPA's air toxics program also includes regulatory and voluntary emission reduction programs that will present many risk management challenges. The technology-based phase of the air toxics program for stationary sources achieves emission reductions through the development of MACT standards. These standards are based on technology already in use by industrial sources. New motor vehicle emission standards for hydrocarbons and particulate matter have resulted in significant decreases in air toxics emissions from mobile sources. Separate standards for motor vehicle fuels have supplemented and enhanced the effectiveness of vehicle controls and led to significant reductions in evaporative and exhaust emissions and specific toxic air pollutants (e.g., benzene, 1,3-butadiene, formaldehyde, acetaldehyde, POM). However, in order to obtain the incremental risk reductions that may be needed to protect public health under EPA's risk-based program, innovative and cost-effective risk management options, including pollution prevention alternatives, should be explored and developed. As a wider array of risk management options becomes available, EPA decision makers will have more

alternatives for targeting risk reductions in areas where risk levels of concern are identified. This strategy includes the research needs to develop these risk management alternatives.

In summary, this strategy is designed to support the air toxics program's changing emphasis to a risk-oriented program. Evaluating and relating risks associated with exposure to air toxics is complicated when attempted with incomplete information. As a result, program offices, like OAR, are required to make decisions under uncertainty, using the best available science and information. While uncertainty may never be eliminated, the goal for ORD's air toxics research is to reduce the uncertainty in assessing and managing the risks associated with exposure to air toxics. This strategy accomplishes that objective through a combination of research that is driven by immediate program activities and longer term research activities.

1.4 RISK ASSESSMENT-RISK MANAGEMENT FRAMEWORK FOR AIR TOXICS

This strategy is organized around the risk assessment-risk management (RA-RM) framework for air toxics (Figure 4) an adaptation of the RA-RM paradigm advocated by the National Academy of Sciences. At the center of this framework are the four major components of the RA-RM paradigm. The outer ring of the framework expands these components into slightly more descriptive elements of the RA-RM paradigm from source characterization to risk reduction. The framework addresses emissions from sources as air toxics enter the atmosphere or the indoor environment and disperse through the environment, with some air toxics undergoing transformation or deposition. Outdoor emissions of air toxics may be found in the ambient air, may be deposited onto water and land, and also may infiltrate indoor environments. Air toxics also may be emitted within indoor environments and, subsequently, may find their way outdoors. Hence, personal exposure may occur through all of these microenvironments and media. The potential for adverse health effects depends on the dose-response relationship of the chemical. Risks are characterized by integrating the exposure and dose-response assessments and by considering the uncertainties and strengths of the evidence. Once exposures and risks are characterized and shown to be unacceptably high, risk reduction methods are needed. After risk management decisions have been implemented, it is important to evaluate whether risk reductions have been achieved and whether the public and environment's health has been

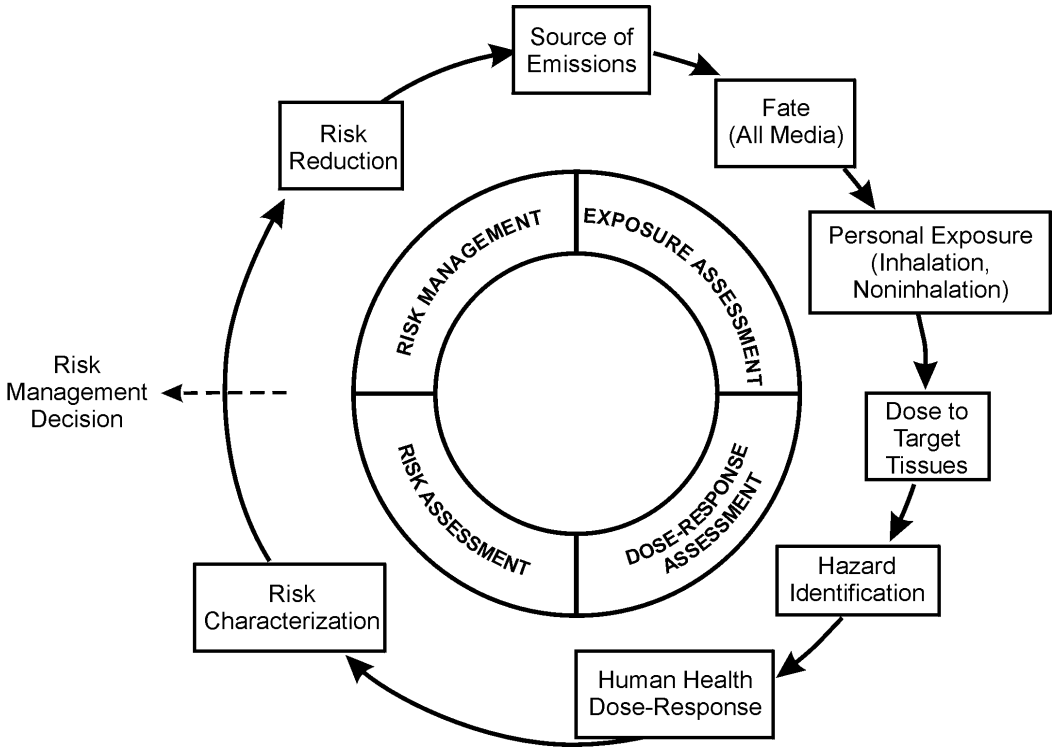

Figure 4. The RA-RM framework.

protected. The following sections provide more detail about each element of the RA-RM paradigm.

1.4.1 Source Characterization

Emission sources for the release of air toxics to the outdoor environment generally can be categorized as described below.

- A *major source*, per Section 112(a) of the CAA, is defined as any stationary source or group of stationary sources (located within a contiguous area and under common control) that emits (or has the potential to emit) 10 tons per year or more of any hazardous pollutant or, in the aggregate, 25 tons per year or more of any combination of air toxics. Large organic chemical manufacturing plants, petroleum refineries, and other industrial operations are typically major sources of air toxics.

- An *area source* is any stationary source that does not qualify as a major source. Dry cleaners, gasoline stations, and chromium electroplating operations are typically area sources of air toxics.
- *Mobile sources* include highway motor vehicles and nonroad mobile sources. Automobiles, buses, aircraft, railroads, construction vehicles, and recreational equipment are considered mobile sources.
- *Indoor air sources* include indoor activities such as cooking and the use of consumer and commercial products, building materials and equipment. Indoor air sources are not subject to regulation under the CAA.

The OAR has developed a National Toxics Inventory (NTI), which includes nationwide emission estimates for all 188 HAPs listed in the CAA. In addition to the NTI, emissions to the outdoor (ambient) environment from many, but not all, major sources of air toxics are compiled by EPA in the Toxic Release Inventory (TRI). But, although the TRI database (U.S. Environmental Protection Agency, 1986b) includes emissions for many large manufacturing sources (which are included in the NTI), it does not include many important sources of toxics emissions, such as mobile sources and many small industrial sources.

The EPA has quantified mobile source emissions in support of a rulemaking under Section 202(l)(1) of the CAA. The *Motor Vehicle-Related Air Toxics Study* (U.S. Environmental Protection Agency, 1993) summarized information on emissions of toxic air pollutants associated with motor vehicles and motor vehicle fuels. Also, those mobile source emissions have been updated to account for new information and recommendations from reviewers (U.S. Environmental Protection Agency, 1999a). These emissions have been included in the NTI.

The EPA also has gained considerable knowledge in indoor air source characterization. The EPA has developed emission testing systems and protocols and emissions modeling for the determination of emissions from consumer and commercial products, building materials, and equipment used indoors.

1.4.2 Fate, Transport, and Transformation

Information pertaining to the fate, transport, and transformation of air toxics is critically important when estimating exposure. Formaldehyde, for example, is not only emitted from sources, but is also formed in the atmosphere. Another example is mercury. The elemental gas

form of mercury found in the atmosphere can travel long distances from the originating source. Other, more reactive forms of atmospheric mercury do not travel very far before depositing to the surface and, thus, present deposition problems that are more local or regional in nature. Models that estimate dispersion of air toxics must account for these types of atmospheric fate, transport, and transformation issues.

If the emissions data from sources (major, area, and mobile) are adequate and, where necessary, the atmospheric transformation products are determined, either dispersion modeling, receptor modeling, or both can be used to estimate the ambient concentration of air toxics. Although dispersion models describe how the emissions mix in the atmosphere and are dispersed, the accuracy of their estimates is limited by, among other things, the resolution of the information in the emission inventory. Another approach, receptor modeling, uses ambient or exposure monitoring data to deduce how much of the pollution comes from each source. This source apportionment approach works best when each source or source category contributes substantially to the total pollution in a unique and distinctive way. In addition to atmospheric fate, transport, and transformation, modeling is needed to understand aquatic and terrestrial fate, transport, and transformation, especially for persistent, bioaccumulating toxics (PBTs).

Ambient air monitoring is an important method of understanding the fate, transport, and transformation of air toxics. Although methods are available for several of the air toxics, some of the methods do not have the sensitivity necessary to measure air toxic concentrations at ambient levels, and others are very expensive to employ.

Air toxics emitted into the indoor environment also undergo transformations. Chemical reactions can occur indoors both through reactions with other chemicals or spontaneously. Air toxics also may be adsorbed onto materials within the environment and later reemitted, can be taken out of the air by particle deposition and later reentrained in the air stream, or can be removed from the indoor environment through active ventilation or exfiltration through the building envelope.

Similarly, chemicals emitted from sources outdoors can make their way into the indoor environment through active ventilation or infiltration mechanisms. The EPA has developed indoor air quality models to estimate the effect of these processes on pollutant levels indoors. Indoor air monitoring data is used to validate these models to ensure that they accurately represent the processes occurring within the indoor environment.

1.4.3 Personal Exposure

Exposure is the contact of an individual with a pollutant at visible external boundaries over time periods. Exposure assessments estimate the frequency, duration, and magnitude of human exposures, as well as the identity of the routes and pathways. Personal exposure data that can be used for direct exposure assessment for air toxics are limited and variable. When personal exposure data gaps exist, either in space or time, models are used to estimate exposure to determine the regulatory impacts on exposure over time. Past exposure studies have demonstrated that, for many volatile air toxics, concentrations in personal air > indoor air > outdoor air. The differences in air concentrations and in the resulting exposure assessments can be substantial, especially for the high-end exposure groups.

Ambient monitoring data and models can be used to assess inhalation exposure if the relationship between these two measures is well understood and has been quantified through well-designed measurement studies. An ambient air monitoring initiative is underway at EPA, in cooperation with state and local agencies (U.S. Environmental Protection Agency, 2000). Data on microenvironmental concentrations, personal exposure concentrations, activity patterns, and other exposure factors (e.g., factors leading to increased susceptibility) first are needed to both develop and evaluate the models. Because people typically spend a large portion of their time indoors, it is also important to understand the indoor/outdoor relationship for air toxics, the factors that affect chemical penetration and decay in buildings, and the important indoor sources of air toxics indoors. In addition, it is important to understand the exposure to air toxics as a result of food being contaminated with deposited air toxics.

Personal exposure monitoring can be used to directly assess exposure. It accounts for the microenvironments where an individual spends time and the pollutant concentrations in those microenvironments. It also accounts for all sources and pathways. Personal monitoring also can provide important information for evaluating multi-route exposures, quantifying exposure variability and high-end exposures, accounting for cumulative exposures (i.e., major source surrounded by area source), accurately measuring exposures for susceptible populations, and defining the factors that are responsible for variability in exposure. It also can be useful in the evaluation of the outputs of exposure models.

Biomarkers measure the air toxic or its metabolite in a biological matrix such as blood, urine, breath, fat, or hair. Biomarkers do not measure directly external exposure, rather they

provide some measure of the pollutant that has entered the body. Although biomarkers do not provide information about the pathway, level, or source of exposure, for some chemicals, biomarkers can serve as a useful measure of direct exposure aggregated over all sources and pathways. Biomarkers also can provide useful information to link exposure, absorbed dose, and target tissue dose.

1.4.4 Target Tissue Dose

Following exposure to air toxics, an adverse effect may occur when the pollutant (or its active metabolite) reaches the target tissue site. This "target tissue" dose is one of the links between exposure and effects. Health effects from exposure to air toxics may be related more directly to the quantitative pattern of deposition in various regions of the respiratory tract than to the actual exposure concentration. Physiologically based pharmacokinetic (PBPK) models permit improved estimation of target tissue concentrations for risk estimation and, for some chemicals and exposure conditions, enable use of oral data to estimate target tissue concentrations relevant to inhalation exposures when effects on the respiratory tract are not a concern. The solubility and reactivity of gases, along with PBPK models, are used by ORD in determining target tissue doses of air toxics as the data allow. The dosimetry of inhaled agents also depends on physicochemical characteristics, such as size and shape of particles that influence aerodynamic behavior and deposition of particles. Thus, both physicochemical and physiologic dosimetric adjustments are used in extrapolating animal data to humans in developing dose-response assessments as part of the risk assessment process used by EPA.

1.4.5 Hazard Identification and Dose-Response Assessment

Dose-response assessment is the process of estimating a response at various applied dose levels. An understanding of underlying mechanisms is a key component in assessing risks posed by exposure to mixtures of chemicals, extrapolating from short- to long-term effects and from animal to human effects, and identifying sensitive subpopulations.

Recent advances in scientific knowledge regarding how exposures to certain air toxics produce cancer and noncancer toxicity, as well as the development of improved computer-based methods to support quantitative modeling of data sets, may result in improved dose-response assessment approaches. These new approaches would reduce uncertainty and provide

harmonization between cancer and noncancer endpoints. Unfortunately, this approach is proving difficult for many air toxics, given the limited availability of information.

1.4.6 Risk Characterization

Risk characterization is the description of the nature and, often, the magnitude of human risk, including attendant uncertainty associated with a risk assessment. Various EPA guidance recommend risk characterizations to have clarity, transparency, reasonableness, and consistency. Risk characterizations are clear if the description and organization of the issues are understandable, the level of effort (quantitative or qualitative) is given, the strengths and limitations of the assessment are delineated, assumptions are described, and complete documentation of the data sources and analytical methods is available. Transparency occurs when conclusions of science and policy are differentiated, alternative interpretations of the data are given, and results of peer review and attendant issues are discussed. A reasonable risk assessment acknowledges the scientific and technical uncertainty and utilizes the best available scientific information and judgment. Risk assessments must be consistent with EPA-wide guidelines regarding accepted indicators of exposure (e.g. daily, lifetime, etc.), dose-response (e.g., cancer unit risk, oral reference dose [RfD], inhalation reference concentration [RfC], etc.), and risk (e.g., probability, hazard quotient).

1.4.7 Risk Management

Risk management research identifies ways to prevent and reduce risks from pollution that threaten human health and the environment. This research includes investigating methods and their cost-effectiveness for prevention and control of ambient and indoor air pollution. The goal of this research is to provide solutions to environmental problems by (1) developing and promoting effective environmental technologies, (2) developing scientific and engineering information to support regulatory and policy decisions, and (3) providing the technical support and information transfer to ensure implementation of environmental regulations and strategies at the national and community levels.

1.5 WHAT IS THE SCOPE OF THIS STRATEGY?

The scope of the air toxics research strategy covers sources of air toxics (stationary, mobile, indoor), multiple routes of exposure, human health effects, and risk assessment and management issues. In some instances, the strategy refers to protecting the environment, as well as, human health. By doing so, the strategy recognizes that air toxics do pose risks to the natural environment and that research efforts are needed to characterize these risks and identify ways to mitigate them. However, based on priorities identified by the Office of Air and Radiation (OAR) and other activities underway in ORD, this strategy does not include any research focused specifically on how air toxic compounds impact ecological systems. Although the priorities for air toxics research are focused on human health, there are likely to be indirect ecological benefits to the extent the research planned leads OAR and the states to adopt human health protection strategies that also reduce deposition to ecosystems. Other ORD strategies include some research to address the ecological impacts of air toxics. For example, the impacts of mercury on sensitive wildlife and research to understand the processes in the natural environment that influence the transformation of mercury into methylmercury (the form responsible for many of the impacts on wildlife and humans) are addressed through the *mercury research strategy*. ORD's *ecosystem research strategy* includes activities to determine the environmental quality of various ecological systems across the country. Levels of a variety of toxic compounds including PAH's, PCB's benzene, lead, mercury and cadmium in sediments and the tissue of various aquatic species are measured through this broad ecosystem. Air deposition is known to contribute to ecosystem loadings. ORD also participates in the Agency's program to address compounds that are persistent and bio-accumulate in the environment (PBT pollutants) such as dioxin and mercury. PBT research includes efforts to understand human and ecological exposure through monitoring studies, efforts to improve estimates from sources such as landfills and open burning, and efforts to developed improved continuous source monitoring techniques. All these programs will generate relevant information to consider when making policy decisions concerning the direction of the air toxics program with respect to ecological issues.

In addition to the ecological research conducted under other ORD strategies, there is also a *human health risk assessment research strategy* that supports development of generic risk assessment methods and other tools and that when completed can be applied to a variety of

Agency programs including air toxics. For example, research is underway to produce a source-to-dose model that will provide the Agency with the capability to connect environmental releases (source) to personal exposures. This strategy also includes efforts to develop improved techniques to assess risks to susceptible sub-populations including children. Once developed, these techniques can be used for future air toxic risk assessments. ORD's pollution prevention and mercury research strategies include research on innovative techniques to reduce and measure air toxic emissions from indoor, stationary and area sources. Efforts are underway to identify novel coating formulations that significantly reduce the content of air toxic pollutants, new techniques to synthesize chemicals (green chemistry) that use less toxic solvents, and technologies to control mercury emissions from utility boilers and other combustion systems. The pollution prevention program also includes ORD's Environmental Technology Verification program that includes several centers to investigate the performance of commercially ready air pollution control technologies and measurement devices including some that address air toxics. Finally, ORD's Small Business Innovative Research Program (SBIR) provides funding for research on novel air toxic control technologies and measurement devices under development in the private sector.

There is also some research that is beyond what ORD can deliver through resources within its centers and laboratories. In these cases, ORD will endeavor to coordinate air toxics research needs with research organizations outside of EPA (such as other federal agencies, private sector groups, academia, and nonprofit research institutions). For example, a critical need for conducting air toxics research is dose-response data. ORD will work with others to supplement its research effort developing dose-response relationships for prototypical chemicals and extrapolating those to other compounds. ORD will also utilize existing information generated by the National Toxicology Program (NTP) and exercise EPA's authority under the Toxics Substances Control Act (TSCA) to have chemical manufacturers provide additional data.

2. THE STRATEGY FOR AIR TOXICS RESEARCH

2.1 INTRODUCTION

This chapter explains the structure and development of the Air Toxics Research Strategy (ATRS):

- use of the air toxics RA-RM framework,
- key overarching research questions organized within that framework,
- summary research needs associated with each key research question,
- the strategic principles used to select priority research, and
- four groups of air toxics that focus air toxics research.

2.2 STRUCTURE OF THE ATRS

The structure of the ATRS is graphically presented in Figure 5. The strategy begins with input about the air toxics program goals and needs, explained generally in Chapter 1. The NATA will develop measures of risk and assessment uncertainty for air toxics and, thus, will provide scientific input for use during implementation of the ATRS. This ensures program relevancy to the science resulting from the ATRS. The air toxics RA-RM framework, as discussed in Chapter 1, was used to organize key research questions and associated research needs. The RA-RM framework presents a logical approach for assessing and managing risks and provides a basis to ensure that a reasonably comprehensive list of relevant research is identified for consideration.

Key research questions were developed considering the current scientific knowledge of air toxics and the air toxics program's goals and needs. These overarching questions are intended as a departure point for developing and organizing more specific research needs. These key questions are presented in Table 2.

The EPA identified research needs that would help answer the key research questions. These research needs were selected to be as comprehensive as practically possible with a special emphasis on filling research gaps, improving our understanding of air toxics for each part of the paradigm, and addressing program goals and needs. Given the comprehensive nature of the

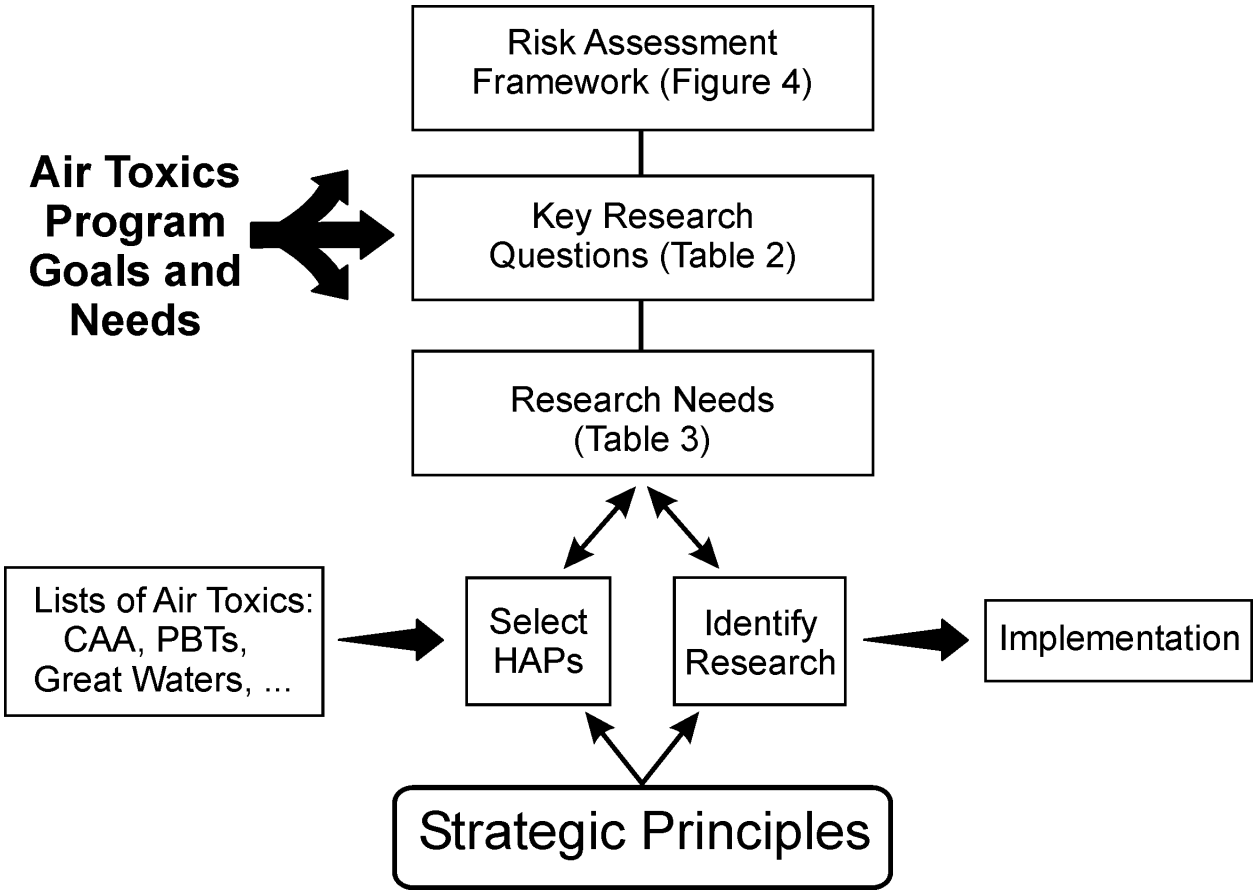

Figure 5. Development of Air Toxics Research Strategy.

TABLE 2. KEY OVERARCHING RESEARCH QUESTIONS FOR AIR TOXICS

1. What are the sources of air toxics and what are their characteristics?
2. What is the role of atmospheric transport, transformation, fate, and chemistry on air toxics concentrations, including indoor, micro-scale, urban, terrestrial, and regional concentrations?
3. What is the relationship of concentrations of air toxics (from outdoor and indoor sources) to personal exposure?
4. What are the health hazards and dose-response relationships associated with exposures to air toxics?
5. What improvements can be made to dose-response assessments?
6. What health risks can be characterized quantitatively for people exposed to air toxics?
7. What risks from air toxics can be prevented and managed cost effectively?

summary research needs, it should be emphasized that not all of this research can be undertaken with current EPA resources.

The ATRS research needs are presented in Table 3 for each key research question. The research needs for each key question are ordered generally consistent with the steps taken during an investigation: defining the question (developing hypotheses), developing methods, gathering data, developing models, performing analyses, and interpreting results.

As explained below, strategic principles were developed to help select air toxics to study, as an initial focus of the ATRS, and to help identify specific priority research for those air toxics. The research needs, identified in Table 3, will be used to develop potential research activities during implementation of the ATRS (e.g., as presented in Chapter 3 for POM and discussed in more detail in Chapter 4).

2.3 STRATEGIC PRINCIPLES

Strategic principles were developed to guide the decisions within the ATRS and to support ORD's priority-setting activities during annual budget and multi-year plan development. As seen in Figure 5, strategic principles (along with the program goals and needs, key research questions, and research needs) influence the selection of air toxics for research. These principles work in tandem in selecting air toxics to study, specific research to undertake, and the priority of that research.

Principle 1. Increase the usefulness of the research program by grouping air toxics initially based on physicochemical properties to assist in future studies of structure-activity relationships (SARs)

The large number of individual air toxics compounds and compound classes makes it impossible to scientifically evaluate each listed air toxic in a reasonable amount of time with the foreseeable resources available. Grouping of compounds within well established chemical classification schemes used in organic chemistry can have long-term and multiple payoffs in terms of linking EPA's research to well established chemical literature on air toxics physical and chemical properties and reactivities that can underlie their biological/nonbiological activities.

TABLE 3. RESEARCH NEEDS

Key Question 1. What are the sources of air toxics, and what are their characteristics?

A. Improve measurement methods for air toxics speciation.

B. Refine current methods and models and initiate the development of new methods and models used to estimate air toxics emissions from indoor sources.

C. Develop and validate measurement methods for industrial and combustion sources of air toxics; develop dilution sampling methods for semivolatile and organic metal species.

D. Develop better emission factors for area and major source categories, including spatial and temporal resolution of emissions for the most important source categories.

E. Improve characterization of air toxics for nonroad vehicles, including emissions test data, and equipment counts and usage to estimate their emission rates.

F. Improve characterization of air toxics for all classes of highway vehicles, including collection of additional emissions test data and consideration of modal emissions variables.

G. Characterize emissions from vehicles operating on new fuels and fuel additives and from prototype low-emitting vehicle and ultra-low-emitting vehicles.

H. Develop data and databases on source emissions, source strengths, and source usage for indoor and other microenvironmental sources.

Key Question 2. What is the role of atmospheric transport, transformation, fate, and chemistry in air toxics concentrations (including indoor, micro-scale, urban, terrestrial, and regional concentrations)?

A. Develop analytic methods for measuring air toxics in microenvironments, in ambient air, and in other environmental media. Develop new methods or improved existing methods to yield better sensitivity and lower detection limits.

B. Carry out laboratory studies to investigate the key chemical and physical processes of volatile, semivolatile, and nonvolatile air toxics that influence (1) ambient concentrations on neighborhood, regional, and global scales, (2) transformation products, and (3) fluxes to land and water bodies.

C. Identify chemical mechanisms or chemical parameters to be incorporated into source-based air quality modeling. Improve the understanding of the limitations of current models, continue developing and refining the models needed to address the movement of air toxics through the environment, and produce modeling tools and techniques that consider the potential multiple pathways and multiple media routes of exposure.

D. Identify, develop data, and model the physical and chemical factors that influence the distribution of air toxics concentrations in residential and nonresidential indoor microenvironments, including penetration factors, air exchange rates, decay rates, removal and sink effects, and source strengths, and usage.

E. Develop chemical and physical modeling approaches that can address the complexities and properties associated with air toxics including, for example, changes in toxicity, congener mix or volatility.

F. Develop methodologies that provide linkages between source-based, urban-to-neighborhood-scale, air quality models for air toxics and exposure models, including determination of temporal and spacial relationships among modeled concentrations and monitoring data.

G. Develop and evaluate source-based multi-scale (regional-to-neighborhood) air quality models, capable of providing acute to long-term chronic exposure time scales.

H. Reconcile source-based modeling with receptor-based modeling to improve the accuracy and performance of each type of approach.

TABLE 3 (cont'd). RESEARCH NEEDS

Key Question 3. What is the relationship of concentrations of air toxics (from outdoor and indoor sources) to personal exposure?

A. Where needed, develop methods for measuring personal exposure for selected air toxics.

B. Identify key outdoor and indoor microenvironments for exposure to air toxics. Evaluate data (relating to exposure) on the sources, concentrations, and variability of air toxics in these key microenvironments. Determine the factors that influence the distribution of air toxics concentrations in these microenvironments and incorporate these into models.

C. Evaluate and, when needed, develop data on ambient, outdoor, indoor, and personal exposure concentrations to air toxics. Determine the relationships between these measures. Determine the factors and data, including population activity patterns, personal activities, and housing characteristics, that influence personal exposures to air toxics. Develop information for the general population and susceptible populations.

D. For relevant pollutants and pathways, develop data and models that estimate multimedia (air, water, food, soil, and dust) and multiroute (inhalation, ingestion, and dermal absorption) exposures to air toxics. For the general population and susceptible populations, characterize the relative contribution of sources, routes, and pathways to total human exposure.

E. Develop data (i.e. respiratory tract deposition and clearance, ingestion rates, dermal absorption, dietary absorption) that allow prediction to dose for the general population and susceptible populations.

F. Evaluate and refine current models (microenvironmental source to dose through multimedia and multi-pathways) to more adequately estimate total human exposures and to identify exposures associated with indoor and outdoor sources of air toxics.

G. Identify and develop the tools needed to characterize the relative contribution of sources, routes, and pathways to aggregate or cumulative exposure and risk, both for the general population and susceptible populations.

H. Develop biomarkers that could be used to measure exposure to air toxics.

Key Question 4. What are the health hazards and dose-response relationships associated with exposure to air toxics?

A. Identify human health outcomes of concern following exposure to air toxics.

B. Develop a better understanding of the importance of exposure scenario in producing health outcomes to include short-term or intermittent exposures versus long-term exposures.

C. Expand the screening methods for hazard identification of health outcomes that are not covered in current Toxic Substances Control Act/Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) test guidelines, but which are of high concern for exposure to air toxic compounds, such as asthma and exacerbation of respiratory diseases.

D. Identify the common modes of action associated with the initiation and development of human diseases for air toxics.

E. Identify mixtures of air toxics compounds for which the hazard may be less than or greater than that predicted by a default assumption of additive toxicity.

F. Identify the relevant factors that increase susceptibility to air toxics.

G. Expand models and data for animal to human extrapolation.

TABLE 3 (cont'd). RESEARCH NEEDS

Key Question 4 (cont'd).

H. Expand models and data for high- to low-dose extrapolations.

I. Expand models and obtain data for extrapolation of health outcome data across exposure routes (especially oral to inhalation).

J. Develop exposure-dose-response models for air toxics compounds that link exposure to internal dose, target tissue dose, and adverse health outcome

K. Identify and develop an understanding of biomarkers and their measure for adverse health outcomes (as a link in the exposure-dose-response continuum).

Key Question 5. What improvements can be made to dose-response assessments?

A. Improve hazard identification and develop cancer dose-response assessments and chronic and acute noncancer dose-response assessments for air toxics.

B. Continue to improve the methods for *acute* noncancer dose-response assessment.

C. Develop improved methods for cancer and *chronic and subchronic* noncancer dose-response assessment.

D. Develop new statistical/analytical approaches to ensure maximum use and interpretation of toxicological data for cancer and noncancer dose-response assessments that include probabilistic dose-response assessment for multi-route and multi-pathway assessments.

Key Question 6. What health risks can be characterized quantitatively for people exposed to air toxics?

A. Develop framework for incorporating mode of action data in risk characterization of air toxics.

B. Improve methods and models for risk characterization of mixtures that incorporate constituent chemical interactions as relevant parameters controlling the risk of air toxics mixtures.

C. Develop methods that enhance quantitative uncertainty analysis to reduce uncertainty in risk characterization.

D. Develop methods for using biomarkers in performing risk assessments.

Key Question 7. What risks from air toxics can be prevented and managed cost effectively?

A. Demonstrate continuous metals monitor for controlled streams for compliance and risk management purposes.

B. Develop cost-effective compliance monitors capable of measuring multiple air toxics.

C. Develop fundamental understanding of how air toxic pollutants are formed or prevented in industrial and combustion processes.

D. Identify processes contributing to the HAP emissions from listed area source categories and listing of control options and pollution prevention (P2) alternatives for these processes.

E. Identify stationary industrial and combustion processes contributing to the air toxics emissions and listing of control/prevention options for these processes.

TABLE 3 (cont'd). RESEARCH NEEDS

Key Question 7 (cont'd). What risks from air toxics can be prevented and managed cost effectively?

F. Evaluate and document additional emissions control and P2 alternatives for area source categories, for toxic-fine particulate control measures, for removal of trace metal air toxics, and for low-concentration/high-volume air toxic emissions/gas streams.

G. Identify P2 alternatives for mobile sources worthy of special studies.

H. Identify P2 alternatives and control technologies appropriate for indoor air sources (e.g., work with industry and other stakeholders to develop lower emitting or lower toxicity products, validate test methods as part of standard-setting activities, create voluntary demand-side market incentive programs).

I. Evaluate the impact of existing and emerging risk management options in reducing risk to indoor air toxics.

J. Develop standardized methods to evaluate the cost effectiveness of indoor air risk management options.

K. Develop an indoor air risk management database of existing and emerging emission control and pollution prevention methods to screen available options for risk management.

L. Characterize the relative ability to manage indoor and outdoor air toxics sources.

Although this principle emphasizes research that produces results with wide applicability, some research on individual air toxics may remain appropriate.

The groupings of air toxics selected for the ATRS are explained in the next section of this chapter. Within groups or classes of chemicals, the study of model compounds should be emphasized as a way to develop information on underlying or fundamental chemical mechanisms and outcomes important to potential human exposure and health. This is similar to the use of 2,3,7,8-tetra chlorodibenzo-*p*-dioxin as a model chemical for the study of the halogenated aromatic hydrocarbons, but extended to include both biological and certain nonbiological activities of importance, such as those controlling environmental fate and distribution and, ultimately, human exposure. The best model chemicals from a scientific standpoint may not always be high on programmatic priority lists, but their study would be justified in terms of their usefulness in providing additional, clarifying information with regard to modes or mechanisms of action of chemicals on the lists.

The ORD has used a set of strategic principles to guide its research planning in recent years (U.S. Environmental Protection Agency, 1997a); the following two of which are especially relevant here.

Principle 2. Focus research and development on the greatest risks to people and the environment.

Principle 3. Focus research on reducing major uncertainties in risk assessment and improving cost effectiveness in risk prevention and management.

These two principles work together in helping identify the greatest need for research (as shown in Figure 6). The result of applying these principles is to direct research towards air toxics that pose the greatest risk and the most uncertainty first. Many air toxics have been associated with serious human effects, although little is understood about the exposures and risks for many of these toxics. As required by the CAA, EPA listed, based on available information, the 33 HAPs contributing to the greatest risks to most people living in urban areas. Recently, EPA identified mobile source air toxics, including diesel exhaust. The NATA initial national-scale assessment shows that these urban HAP emissions and ambient concentrations occur in essentially all census tracts throughout the United States. The EPA has identified several air toxics that are important for exposures indoors. In addition, several studies have culminated in identifying about one dozen air toxics of particular concern for the Great Waters. Appendix B provides a summary of the human health concerns for many air toxics.

As an indication of the uncertainty associated with each of the air toxics, an evaluation (see Appendix B) was made of the presence or absence of cancer unit risk, RfC, and RfD values for each listed air toxic, and, if RfC or RfD values were available, the size of the associated uncertainty factors. In this evaluation, it was assumed that air toxics without cancer unit risk, RfC, or RfD values have greater uncertainty associated with their risk assessment than those with listed values, that high uncertainty factors reflect greater hazard uncertainty associated with their risk assessment than those with listed values, and that high uncertainty factors reflect greater uncertainty than low values. Although this evaluation does not address chemical exposures or cumulative and aggregate exposures and risks, it can provide a useful initial emphasis. As NATA activities are evaluated, ORD will use the results to provide a basis for further indications of uncertainty.

In addition to the two most relevant ORD strategic principles, two additional strategic principles were developed for use in the ATRS. These additional principles are intended to ensure ORD's air toxics research supports the air toxics program in an efficient manner.

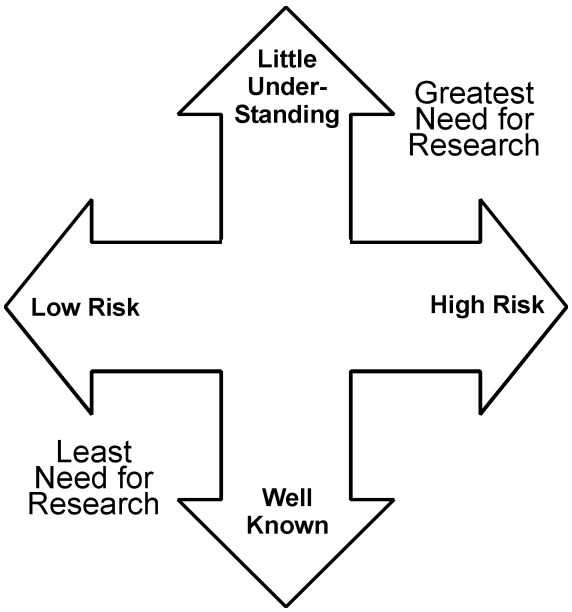

Figure 6. Determination of research needs with regard to risk and uncertainty.
(Adapted from Paul Slovic, *Risk Perception*.)

Principle 4. Undertake and foster multidisciplinary research.

Under this operational principle, ORD would emphasize research that can provide complete risk assessment and risk management solutions by integrating research across scientific disciplines. Within the context of the RA-RM framework, this principle helps to identify research that motivates scientists to develop trans-disciplinary research and communications. This principle encourages research relevant to multi-pollutant and multi-pathway exposures and aggregate risks and emphasizes the value and need for "leveraging" across research in other program areas (e.g., particle research may provide useful information for air toxics).

Principle 5. Ensuring an appropriate balance between near-term research and long-term research.

Important, time sensitive research is needed to underpin the air toxics program now. In addition, long-term research should be completed over time to support key emerging air toxics programmatic objectives. Accordingly, ensuring an appropriate balance between near-term and

long-term air toxics research is essential. Near term research would be conducted such that results would be available for use within the air toxics program within 5 years, with long-term research available within roughly 10 years. In addition, it is also important to consider that some research projects must be sequenced properly, if the completion of one project is critical to the conduct of another. That is, some long-term research must begin in the near term and this should be reflected in the prioritization process. For example, if research into short-term, high volume emission and exposure scenarios, along with research into acute health effects for the appropriate air toxics, had begun when the CAA was amended in 1990, the NATA would perhaps be able to provide a national assessment for such situations at this time.

In summary, the ATRS is primarily centered around the first principle. That is, the first step in setting priorities is to identify HAP groups to study. In selecting the HAP groups, it is important to consider the second and third principles; that is, HAP groups should be selected representing the greatest risks and most uncertainty. Then, using the fourth and fifth principles, multi-disciplinary research should be emphasized, with a balance between near-term and long-term research. As such, the ATRS focuses on groups of important air toxics, while being sufficiently flexible to adequately respond to change, in response to the multitude of air toxics with their effect, exposure, risk, and management issues. This especially is needed when aggregate and cumulative exposures and risks must be considered. The ORD and ATRS-specific operating principles will help with specific air toxics research plans that will result from periodic scientist-to-scientist meetings (see Chapter 4), in which NATA results will help define the risks and uncertainties of concern. Accordingly, the ATRS provides *guidance* for an integrated and scientifically credible research program.

2.4 SELECTION OF AIR TOXICS GROUPS AND POTENTIAL PRIORITY HAPs

One task associated with air toxics research is the prioritization of chemicals to be studied. Because study of every air toxic pollutant is impractical within a reasonable time frame, it is highly desirable to develop knowledge about a given air toxic pollutant that can be used credibly to inform decisions about other air toxics (with related properties). The ability to use information in this manner may vary by the portion of the RA-RM framework (see Figure 3, Chapter 1) in which the grouped air toxics are being studied.

There are different ways that air toxic chemicals can be grouped. The structure of chemicals and their associated physical and chemical properties and reactivities ultimately determine their behavior with respect to emission characteristics, fate and distribution, exposure and bioavailability, dose-response, and effects. As a result, one possibility for grouping HAPs is to do so by a chemical structure. Another possibility is to group air toxics based on the greatest health risk (highest exposure and highest toxicity) and the greatest uncertainty. Yet another possibility, is to group chemicals based on the types of sources from which they are emitted or by the environments in which the chemicals are commonly found.

This section starts with a discussion of factors that are relevant to the different elements of the RA-RM framework which should be considered when grouping HAPs. The section then goes on to present different approaches for grouping HAPs and ends with a crosswalk of the different approaches to yield a potential list of priority HAPs.

2.4.1 Factors Relevant to Exposure, Health, and Risk Characterization and Management

With respect to exposure, exposure assessments for air toxics have focused on inhalation as the primary route of exposure. However, ingestion and dermal absorption may be important routes for those chemicals that are less volatile and more hydrophobic. For these chemicals, multimedia, multi-pathway, aggregate exposure assessments must be conducted to fully characterize both exposure and risk. The EPA's long-term goal is to characterize aggregate exposures to air toxics and to quantify cumulative exposure and risk from mixtures of chemicals. The chemical mixtures from sources are the mixtures that are most important for cumulative exposure. Thus, as EPA moves toward research on cumulative exposures, selection of chemical groups and priority chemicals within the groups may be based on source mixtures rather than chemical structure.

With respect to health effects, structural and chemical reactivity considerations were useful in meeting the objective of selecting smaller sets of relevant chemicals for study based on mode or mechanism of action, chemical class, or other proximity indicators that would improve the predictive capabilities of SAR models. In addition to physical/chemical and structural/reactivity families, other approaches to classification include mechanistic families (alkylating/arylating/ acylating reagents, etc.). These may evolve as more is learned about the mode of action of selected chemicals. For the chemicals with more complex structures with several functional

groups interacting, with tautomeric and conformational changes, or ionizations occurring near physiological pH, etc., more specific effects on a particular organ/tissue/cellular system are anticipated, and thus they may be more difficult to model and be less useful as prototype chemicals for groups. The selected HAP groups provide a framework for initiating work and then recognizing this potential problem area.

This approach results in a minimum number of groups based on structural chemistry, and reactivity classifications were identified based on the current knowledge about chemical mechanisms underlying toxicity (see Appendix B). Such a process also may facilitate selection of prototype chemicals that could be studied in a mechanistic framework across the various programs and, ultimately, provide information and understanding that could be applied through SAR to other air toxic chemicals. Furthermore, the identification of fundamental chemical mechanisms that underlie both biological and nonbiological activities can help unify different program interests and objectives. A potentially unique objective of the proposed approach is to identify as many of these common threads as possible and build on them. Further analysis and consultation, as the ATRS is implemented, may yield a more refined chemical classification scheme that will be more likely to identify sets of air toxics sharing common hazard profiles.

To conduct health risk assessments as outlined in Chapter 1, dose response information is necessary. Using the combined list of air toxics, the Integrated Risk Information System (IRIS) database was searched for the availability of cancer unit risk factors, RfCs, and RfDs. There were 36 cancer unit risk factors, 34 RfCs, and 59 RfDs on IRIS for the 188 CAAA-listed HAPs. Some IRIS assessments were available but had high uncertainty factors (>1,000) and thus also point to the need for improved dose-response data and further dose-response assessment development. Current assessment activities in ORD also were sought out to determine those cancer and noncancer dose-response assessments underway. A list of air toxics and the status of dose-response development are included in Appendix B.

From a risk management point of view HAP groups are best defined by the sources of the air toxics which are emitted because these sources will be the subject of risk management decisions and research.

2.4.2 Grouping HAPs by Chemical Structure

Some properties and reactivities are more important than others in determining certain actions. In the absence of better information, a preliminary chemical structure/reactivity classification approach was used to divide the list of air toxics into 11 classifications, including the (1) alcohols/glycols, (2) aldehydes/ketones/other potential acylating agents, (3) alkylators (epoxides, aziridines, and others), (4) amines/precursors/derivatives, (5) ethers/alkyl and aryl, (6) halides/alkyl and aryl, (7) hydrocarbons/aromatic and nonaromatic, (8) metals/minerals and other inorganics, (9) organic acids/precursors/derivatives, (10) phenols and derivatives, and (11) sulfur/phosphorus-containing compounds (see Keith and Walker, 1995). In this approach, some chemicals can appear in more than one group when they have multiple chemical functionality and associated properties. It is presumed that more relevant and specific classification approaches may be developed as the appropriate properties and questions of interest are identified for air toxics.

This chemical structure/reactivity classification approach provides a beginning framework from which to develop studies aimed at linking mechanistically-defined environmental/biological actions with chemical structure and specific molecular triggering events. From this preliminary grouping, the following four HAP groups are suggested as focal points for air toxics research: (1) halides, (2) aldehydes, (3) metals, and (4) POM/hydrocarbons. These four groups (see Table 4) contain many of the compounds of interest for key EPA air toxics programs and represent areas of uncertainty within the key research questions and research needs. Appendix B provides further information on the four HAP groups.

The group of POM/hydrocarbons is associated with urban air toxics, mobile source emissions, and Great Waters and is implicated in residual risk evaluations. As shown in Appendix B, there are many uncertainties that are reflected in the summary research needs discussed under each overarching research question. Although many physical properties are understood from an exposure perspective for hydrocarbons and POM, EPA lacks dose-response assessment values for the many POM and additional exposure relevant information for the entire group to use in a formal risk assessment. Thus, ATRS research into hydrocarbons and POMs would be program relevant and address key research questions and needs.

There are 11 air toxics in the aldehyde group. As shown in Appendix B, three of the aldehyde air toxics are implicated in the residual risk program and for urban air toxics (area and

TABLE 4. AIR TOXICS WITHIN THE ATRS AIR TOXICS GROUPS

Aldehydes and Ketones/Acylating Agents	
Acetaldehyde	75-07-0
Acetophenone	98-86-2
Acrolein	107-02-8
Butanone, 2-	78-93-3
Chloroacetophenone	532-27-4
Formaldehyde	50-00-0
Hydroquinone	123-31-9
Isophorone	78-59-1
Methyl-2-Pentanone, 4-	108-10-1
Propionaldehyde	123-38-6
Quinone	106-51-4

Metal/Minerals	
Antimony Trioxide	1309-64-4
Arsenic +3	7440-38-2+3
Arsenic +5	7440-38-2+5
Asbestos	1332-21-4
Attapulgite	12174-11-7
Cadmium Compounds	7440-43-9
Beryllium Compounds	7440-41-7
Chromium +6	7440-47-3+6
Chromium +3	7440-47-3+3
Erionite	12510-42-8
Hydrochloric Acid	7647-01-0
Lead	7439-92-1
Manganese Compounds	7439-96-5
Mercuric Chloride	7487-94-7
Nickel	7440-02-0
Nickel Subsulfides	12035-72-2
Nickel Chloride	7718-54-9
Nickel Sulfate	7786-81-4
Selenic Acid	7783-08-6
Selenious Acid	7783-00-8
Selenium Dioxide	7446-08-4
Selenium	7782-49-2
Silica	14808-60-7
Sodium Selenide	1313-85-5
Sodium Selenate	13410-01-0
Sodium Selenite	10102-18-8
Talc	14807-96-6

Polycyclic Organic Matter (POM)[b]/Hydrocarbons[a]	
Benzo[*a*]anthracene	56-55-3
Benzo[*a*]pyrene	50-32-8
Benzo[*b*]fluoranthene	205-99-2
Benzo[*k*]fluoranthene	207-08-9
Chrysene	218-01-9
Dibenzo[*a,h*]anthracene	53-70-3
Indeno[1,2,3-*cd*]pyrene	193-39-5
Benzene	71-43-2
Biphenyl	92-52-4
Butadiene, 1,3-	106-99-0
Ethylbenzene	100-41-4
Hexane	110-54-3
Naphthalene	91-20-3
Styrene	100-42-5
Toluene	108-88-3
Trimethylpentane, 2,2,4-	540-84-1
Xylenes	1330-20-7
Diesel exhaust[b]	

Halides	
2,4-D	94-75-7
Allyl Chloride	107-05-1
Benzotrichloride	98-07-7
Benzyl Chloride	100-44-7
Bis(2-Chloroethyl) Ether	111-44-4
Bischloromethyl Ether	542-88-1
Bromoform	75-25-2
Carbon Tetrachloride	56-23-5
Chlordane	57-74-9
Chloroacetic Acid	79-11-8
Chlorobenzene	108-90-7
Chlorobenzilate	510-15-6
Chloroethane	75-00-3
Chloroform	67-66-3
Chloroprene	126-99-8
DDE, P,P'-	72-55-9
Dibromochloropropane	96-12-8
Dichlorobenzene, P-	106-46-7
Dichlorobenzidine, 3,3'-	91-94-1
Dichloroethane, 1,1-	75-34-3
Dichloroethane, 1,2-	107-06-2
Dichloromethane	75-09-2
Dichloropropane, 1,2-	78-87-5
Dichloropropene, 1,3-	542-75-6
Dichlorvos	62-73-7
Ethylene Dibromide	106-93-4
Heptachlor	76-44-8
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-68-3
Hexachlorocyclopentadiene	77-47-4
Hexachloroethane	67-72-1
Lindane	58-89-9
Methyl Chloride	74-87-3
Methyl Bromide	74-83-9
Methyl Iodide	74-88-4
Pentachlorophenol	87-86-5
Polychlorinated Biphenyls	1336-36-3
TCDD, 2,3,7,8-	1746-01-6
Tetrachloroethane, 1,1,2,2-	79-34-5
Tetrachloroethylene	127-18-4
Toxaphene	8001-35-2
Trichlorobenzene, 1,2,4-	120-82-1
Trichloroethane, 1,1,1-	71-55-6
Trichloroethane, 1,1,2-	79-00-5
Trichloroethylene	79-01-6
Trichlorophenol, 2,4,6-	88-06-2
Trichlorophenol, 2,4,5-	95-95-4
Vinyl Chloride	75-01-4
Vinyl Bromide	593-60-2
Vinylidene Chloride	75-35-4

[a]Representative PAHs are given

[b]EPA is interested in diesel exhaust as the total particle which may include POMs and hydrocarbons because of its potential significant health risks.

mobile source emissions), whereas two of them are objectives of the indoor program. In addition, one of these three aldehydes does not have a cancer unit risk estimate, and the RfCs for all three are relatively uncertain.

The halides group contains 51 air toxics, 11 of which are urban air toxics, four are of interest for the indoor air program, and five are Great Waters pollutants. Several of these air toxics are implicated in residual risk evaluations. Of the halide urban and residual risk air toxics, four do not have cancer unit risk estimates. Many of the remaining halides are emitted, and research is needed to improve the ability to assess the effects, exposures, and risks associated with this group.

There are 29 air toxics in the metals group, with 12 urban and mobile air toxics, all associated with the residual risk program, and one HAP of particular interest to the indoor air program. Of these air toxics, four do not have needed dose-response assessment values.

2.4.3 Priority HAPs by Program Objectives

The EPA's Air Toxics Program addresses emissions from both stationary and mobile sources, as well as, concentrations found in indoor environments. As a result, there are numerous hazardous air pollutants (HAPs) which could be the focus of EPA research. The 1990 Clean Air Act Amendments identified 188 pollutants as hazardous air pollutants (HAPs) and this list of pollutants is referred to as the list of "air toxics." However, as mentioned previously, additional chemicals and chemical mixtures that may present risks to humans or the environment may also be considered air toxics. As a result, the number of HAPs that could be considered air toxics is quite large, which presents a problem when trying to focus research efforts and has, to an extent, hindered progress in EPA Air Toxics Research program.

The problem of identifying HAPs upon which to focus was not unique to EPA's ORD. This problem was also faced by the EPA's Office of Air and Radiation (OAR). To combat this problem and to provide focus for its air toxics assessment (e.g., NATA), stationary, mobile, and indoor air programs, the OAR has established lists of HAPs which are relevant to specific programmatic objectives. These lists were derived from screening analyses which considered the potential risks of HAPs emitted from different sources (area, stationary and mobile) and found in different environments (urban areas and indoor). While the analyses used to construct these lists contained significant uncertainties and gaps, the resulting lists of HAPs provide

valuable information useful for prioritizing HAPs that are particularly important to different aspects of EPA's air toxics program.

Table 5 presents the Air Toxics Assessment HAPs. This list is more commonly referred to as the Urban HAP List or the "Dirty 33" and was first published in the EPA's Integrated Urban Air Toxics Strategy (U.S. EPA, 2000b) in July of 1999. However, for the purposes of this Air Toxics Research Strategy, this list is identified as the Air Toxics Assessment HAPs because these HAPs have been the focus of the air toxic assessments conducted under the NATA program which this Strategy will support by improving the underlying science.

TABLE 5. AIR TOXICS ASSESSMENT HAPs[a]

Acetaldehyde	Coke oven emissions[b]	Mercury Compounds
Acrolein[b]	1,3-Dichloropropene	Methylene Chloride
Acrylonitrile	Dioxin	Nickel Compounds
Arsenic Compounds[b]	Ethylene Dibromide	Perchloroethylene
Benzene[b]	Ethylene Dichloride	Polychlorinated biphenyls
Beryllium Compounds	Ethylene Oxide[b]	Polycyclic Organic Matter[b]
1,3-Butadiene	Formaldehyde[b]	Propylene Dichloride
Cadmium Compounds	Hexachlorobenzene	Quinoline
Carbon Tetrachloride[b]	Hydrazine[b]	1,1,2,2-Tetrachloroethane
Chloroform	Lead Compounds	Trichloroethylene
Chromium Compounds[b]	Manganese Compounds[b]	Vinyl chloride

[a]This list is also referred to as the Urban HAP List
[b]This HAP was identified as a national or regional risk driver in the 1996 NATA National Scale Assessment, which recognizes significant uncertainty in risk values calculated therein.

The Mobile Source Air Toxics list is presented in Table 6. This list was included as part of the EPA Mobile Source Air Toxics Rules (U.S. EPA, 2001) and provides the air toxics that are commonly known to be emitted from mobile sources and to pose a potential health risk.

Table 7 lists the HAPs that are commonly found in indoor environments at levels which are suspected to pose health risks (EH&E, 2000). This list was drawn from the Indoor Air Toxics Strategy, which is currently under Science Advisory Board (SAB) review and expected to be finalized in 2002.

TABLE 6. MOBILE SOURCE AIR TOXICS

Acetaldehyde	Dioxin/furans	MTBE
Acrolein	Ethylbenzene	Naphthalene
Arsenic Compounds	Formaldehyde	Nickel Compounds
Benzene	n-Hexane	Polycyclic Organic Matter
1,3-Butadiene	Lead Compounds	Styrene
Chromium Compounds	Manganese Compounds	Toluene
Diesel Particulate + Diesel Exhaust Organic Gases	Mercury Compounds	Xylene

TABLE 7. INDOOR AIR TOXICS

Acetaldehyde	Carbon Tetrachloride	Formaldehyde
Aldrin	Chlordane	Heptachlor
Arsenic	Chloroform	Methyl Chloride
Benzene	1,4-Dichlorobenzene	Methylene Chloride
alpha-BHC	Dichlorvos	Perchloroethylene
gamma-BHC	Dieldrin	Trichloroethylene

As described earlier, the EPA's major source stationary source air toxics program contains two phases. The first phase is to develop standards based on technology that is commonly used. These standards are referred to as Maximum Achievable Control Technology (MACT) standards. The second phase of the program is to consider the risk that remains, i.e., residual risk, after implementation of the MACT standards. These "residual risk assessments" are required within 8 years of promulgation of each MACT standard developed. As result, the first residual risk assessments will be conducted for the initial sets of MACT standards to be promulgated. Eventually, the residual risk assessments will address all of the 188 HAPs. However, there are certain HAPs which were addressed by the initial MACT standards and thus, will be the subject of the early residual risk standards. Table 8 lists these HAPs.

TABLE 8. STATIONARY SOURCE AIR TOXICS SUBJECT TO EARLY RESIDUAL RISK STANDARDS

Acetaldehyde	Dioxane	Methyl Ethyl Ketone
Acrolein	Dioxins	Methyl Isobutyl Ketone
Acrylonitrile	Epichlorohydrin	Methylene Chloride
Antimony	Ethylene Dibromide	Naphthalene
Arsenic	Ethylene Dichloride	Nickel Compounds
1,3-Butadiene	Ethyleneglycol	2-Nitropropane
Benzene	Ethylene Oxide	PAHs
Cadmium Compounds	Formaldehyde	Perchloroethylene
Chlorine	Furanes	Phenol
Chloroform	Glycol Ethers	Styrene
Chloroprene	Hexane	Toluene
Chromium Compounds	Hydrochloric Acid	Toluenediisocyanate
Cobalt	Hydrocyanic Acid	1,1,1-Trichloroethane
Cresol	Lead Compounds	1,1,2-Trichloroethane
Cumene	Manganese Compounds	Trichloroethylene
Dibenzofurans	Mercury Compounds	Vinyl Acetate
Dibutylphthalate	Methanol	Xylene
Dichloroethyl ether	Methyl Chloride	

while focused on HAPs with available data, includes enough flexibility to also select chemicals with knowledge gaps in regard to emissions, exposure, health effects, risk characterization, or risk management.

2.4.4 Identification of Priority HAPs

The previous sections have presented different ways to priortize air toxics. Each approach has merit and brings relevant factors into consideration. Table 9 presents a crosswalk of the two approaches discussed in the previous two sub-sections. Specifically, this table shows the HAPs which are in one of the chemical structure groups and are also identified as a priority by at least one of the Program objectives (Tables 5-8). Table 9 can be used to identify HAPs upon which to focus research efforts. However, it is also important to remember that the information used to

TABLE 9. CROSSWALK OF CHEMICAL STRUCTURE GROUPS AND PRIORITY PROGRAM AIR TOXICS

Chemical Structure HAP Groups			
Aldehydes/ Ketones	**Metals**	**POM/ Hydrocarbons**	**Halides**
Acetaldehye (4)	Arsenic Compounds * (4)	Benzene* (4)	Chloroform (3)
Formaldehyde* (4)	Chromium Compounds* (3)	1,3-Butadiene (3)	Dioxin (3)
Acrolein* (3)	Lead (3)	POM/PAH* (3)	Perchloroethylene (3)
Methyl Ethyl Ketone	Manganese Compounds* (3)	Hexane (2)	Trichloroethylene (3)
Methyl Isobutyl Ketone	Mercury Compounds (3)	Naphthalene (2)	Carbon Tetrachloride* (2)
	Nickel Compounds (3)	Styrene (2)	Ethylene Dibromide (2)
	Cadmium Compounds (2)	Toluene (2)	Methyl Chloride (2)
	Antimony	Xylene (2)	Methylene Chloride (2)
	Beryllium Compounds	Cumene	Chlordane
	Cobalt	Dibenzofurans	Chlorine
		Diesel Exhaust	Chloroprene
		Ethylbenzene	1,4-Dichlorobenzene
			1,3-Dichloropropene
			Dichlorvos
			Epichlorohydrin
			Ethylene Dichloride
			Heptachlor
			Hexachlorobenzene
			PCBs
			1,1,2,2-Tetrachloroethane
			1,1,1-Trichloroethane
			1,1,2-Trichloroethane
			Vinyl Chloride

Note - the number in parenthesis (X) indicates that the chemical appears in multiple program objective HAP groups, and the * indicates suggestion that the particular HAP could be a national or regional driver of carcinogenic risk or non-carcinogenic hazard according to the 1996 National Scale Assessment (http://www.epa.gov/ttnaw01/sab/06-sab-nata-risk.pdf). Ethylene oxide, coke oven emissions, and hydrazine were also suggested as national or regional risk drivers.

develop groups based on similar chemical structure and the information upon which Program objective HAP groups are based contain uncertainties. In addition, there are a number of other compounds found in both indoors and ambient environments that could not be evaluated due to a lack of health data. These uncertainties and limitations require a prioritization method that,

3. THE STRATEGIC APPROACH APPLIED TO POLYCYCLIC ORGANIC MATTER

3.1 INTRODUCTION

The immediate practical consequence of the ATRS is to lay out a direction for research for the four groups of air toxics chosen ([1] aldehydes and ketones, [2] halides, [3] metals, and [4] POMs and hydrocarbons). It is believed that research on particular chemicals within a group will facilitate understanding of the entire group. As an example, this section describes the application of the strategy's direction to research for POMs, which will result in a greater understanding of mode of action, dose-response, exposure, and risk characterization of air toxics from many sources. The POMs were selected arbitrarily for this example; this choice does not indicate a priority over the other three groups. As discussed further in the next chapter, future scientist-to-scientist meetings will develop a research priority for the other groups.

Polycyclic organic matter is defined in the CAA Section 112(b) as including compounds with more than one benzene ring and which have a boiling point greater than or equal to 100 °C. For the great majority of sources listed in the NTI to be controlled by the CAA, POMs generally can be thought of as polycyclic aromatic hydrocarbons (PAHs). The *Integrated Urban Strategy* has identified seven PAHs as surrogates for POM. These include 4-ringed (benzo[a]anthracene, and chrysene), 5-ringed (benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, and dibenzo[a,h]anthracene), and 6-ringed (indeno[1,2,3-cd]pyrene) PAHs.

3.2 PRIORITY RESEARCH FOR POM

Priority research is developed for the air toxics groups through the ATRS by considering the research needs for each key question and the strategic principles. For example, priority research for POM would be associated with interdisciplinary research and include a balance between near-term and long-term efforts. Some longer term research also would be considered a priority because it would be necessary to begin such work now in order to achieve results needed in the future. In this section, priority research is presented for POM based on the key questions.

The potential research area is labeled according to the key question and research need found in Table 3. Correlation with research needs is noted in brackets.

Key Question 1: What are the sources of air toxics, and what are their characteristics?

Most POM/PAH are formed during combustion. Major outdoor sources include automobile and diesel exhaust, wood burning, industrial combustion, and commercial charcoal grilling. Important indoor sources include smoking, wood burning, gas heating, cooking, and barbecuing. To support risk assessments such as those conducted as a part of NATA, improved emissions data are needed. In the near term, these needs focus on area and combustion sources. The emissions data include estimates of the quantities of emissions of POM/PAH collectively and for individual pollutant species. Information to quantify the temporal and spatial distribution of these emissions also is required. Source characteristics, such as emission-producing process identifiers, design capacities, material through-puts, and physical stack parameters for major sources also are included. These data support other risk assessment activities as inputs to ambient and indoor air dispersion models, exposure models, and assessments for risk management options. Emission factors for PAH/POM are based on limited or outdated information, and are of poor quality. A critical near-term need is to improve emission factors for area source categories to reduce uncertainty in NATA activities. There is also a near-term need to improve the methodologies for predicting the spatial and temporal patterns of these emissions. To accomplish these objectives, research is needed to develop improved emission factors and spatial/temporal resolution techniques for PAH/POM emissions from boilers, stationary internal combustion engines, waste incineration, residential wood combustion, open burning sources (forest wildfires and prescribed burning, and burning of scrap tires), cyclic crude and organic chemical production processes, and gasoline distribution operations. There is a need to perform source measurements to produce data to improve emission factors and source activity data that would be used to assess surrogate factors for activity. Also, the use of ambient-based methods, including source receptor modeling, and other nontraditional approaches would be investigated to estimate the uncertainties in estimated emissions. [1A]

For some industrial and combustion sources, there is a near-term need for improved source sampling methods. Research to develop the application of innovative continuous emissions monitors (CEMs), such as Jet-resonance enhanced multiphoton ionization (REMPI), and on-line

gas chromatography for use as a system diagnostic tool, MACT screening tool, and research tool would be conducted. [1C]

As is the case for area sources, emission factors and temporal/spatial resolution methods for nonroad mobile sources need improvement in the near term. Emissions tests on gasoline and diesel engines used in nonroad equipment would be performed. [1E]

For highway vehicles, more on-road and laboratory dynamometer emissions tests of gasoline and diesel engine vehicles are needed in the near term. Emphasis should be on determining total and modal emission rates of POM/PAH from diesel/diesel hybrid engines. Heavy truck activity data are needed to improve methods for modeling of spatial and temporal activity patterns of this sub-fleet. Ambient/receptor modeling studies would be performed to validate emissions models, and to assess the uncertainty of estimated emissions for all classes of highway vehicles. [1F,G]

In conjunction with the source measurement activities listed above, sampling protocols for speciation of POM/PAH emitted as gases and particulate matter would be developed and employed for emissions tests to produce speciated emissions data. [1D]

Although limited data are available on source strengths from combustion processes within homes, there are no or little data for other indoor environments. Research needs to focus on anticipated POM sources that have not been characterized adequately for nonresidential buildings (e.g., restaurants). Indoor air source characterization methods and models need to be developed to estimate emissions from these environments. These data should be incorporated into a general purpose database containing emissions estimates for indoor sources. [1B,H]

Key Question 2: What are the roles of transport, fate, and chemistry on air toxics concentrations?

Ambient Air Modeling: To complement and augment the ambient monitoring data, emissions-based air quality models are needed to provide information on the ambient spatial and temporal distribution of air toxics under a wide range of meteorological conditions and for large and varied geographic areas. Exchanges between ambient air and various microenvironments are needed for human exposure models. The current Models-3/Community Multiscale Air Quality (CMAQ) modeling system represents the state-of-science simulation capability for photochemical oxidants, fine and coarse particle matter, and deposition of acidic and nutrient species. Further research would extend CMAQ's capability to POM/PAHs.

Model development would be conducted as described below.

- Incorporate laboratory research outputs into models. [2C,E, near-term need] Laboratory research would provide a basis for modeling processes such as photolyses and chemical transformations, by-products from these transformation, and chemo-physical properties that control sorption onto particulate matter. The parameterizations may include new chemical mechanisms that handle the interactions between PAHs and short-lived radical species in the atmosphere.
- Develop and test nested-grid resolutions that adequately compute the spatial distribution of pollutant concentrations at neighborhood scales. [2F, near-term need] Additional parameterizations may be needed at the finer resolution. For example, at neighborhood scales, methods are needed to transport pollutants through urban canopies, to link transport between subgrid to grid scales, and to exchange pollutants between outdoor and indoor environments.
- Develop methods that use model results and monitoring data. [2C,F, near-term need] Monitoring data provide a means for evaluating models. Fusion techniques for combining monitoring data with model results would enhance model credibility, as well as provide more detailed information than monitoring data alone. Combining modeling and monitoring data also provides a relatively fast and powerful means to enhance exposure models that conduct assessments and implement regulations.
- Develop methods for aggregating modeling at episodic scale to longer term exposure time scale. [2G] Methods would be developed to provide commensurate data for longer term analyses and assessments, as well as for use with human exposure models based on probabilistic paradigms.

Indoor Air Modeling [2D, near-term needs]: As for ambient air, indoor air models are important in estimating the exposure concentrations that may result from both outdoor and indoor emissions sources. In addition, direct measurements of pollutant levels indoors are necessary to validate these models to ensure that they accurately portray the exposure concentrations that may result from these emissions. Indoor air quality models take into account how a pollutant enters the indoor microenvironment (through an indoor emissions source or through direct ventilation or air infiltration), how the pollutant moves through the microenvironment (through air movement, deposition/reentrainment, adsorption/desorption, air filtration, etc.), how it may be chemically transformed within the microenvironment, and how it leaves the microenvironment (through direct ventilation or exfiltration). Current indoor air

models would be adapted for POM. These models would address sources of POM indoors, penetration factors for POM from outdoors, chemical transformations, sink effects, the effects of human activities, etc.

Chemistry [2B, near-term needs]: Research would be carried out to fill in information gaps in the chemistry modules contained in air quality models used to predict ambient concentrations and deposition fluxes of POM. Research also would be carried out to assess the chemical interactions of POM in the indoor environment, so that these effects can be incorporated into indoor air models. Many of the same chemical processes of interest in the outdoor environment can occur indoors as well. However, the mixtures of chemicals occurring indoors may vary somewhat from those found outdoors, because of indoor sources, the lack of photolysis reactions, etc.

The first phase of the research would consist of conducting a detailed evaluation of how chemical and deposition processes of PAHs are parameterized in present models and critically analyzing the laboratory and field data employed to develop the parameterizations. Based on these findings, a laboratory program would be designed to fill in the information gaps. The laboratory results would then be used to develop chemistry modules for incorporation into models.

While the specifics of the laboratory program await completion of the critical evaluation, it is likely that the research would focus on investigating the homogenous and heterogeneous chemical processes that control the lifetimes of the PAHs. Because some of the PAHs will partition between the gas and aerosol phases, partitioning coefficients would be determined as a function of temperature, relative humidity, aerosol chemical composition, and aerosol phase (solid versus liquid). These same parameters are likely to affect a variety of chemical transformations that nonvolatile PAHs, bound to aerosols, may undergo, including photolysis, ozonolysis reactions, and reactions with nitrogen dioxide, nitric acid, and nitrogen pentoxide. Additional kinetic experiments may be required to measure photolysis rates of the gas-phase component of semivolatile PAHs and their gas-phase reaction rate constants with hydroxyl radicals, ozone, and nitrate. Product studies would be conducted to determine the fate of PAHs that undergo gas-phase reactions or reactions on or within the aerosol. The importance of atmospheric transformations of PAHs has been demonstrated by studies that show production through atmospheric transformations is the largest source of nitro-PAHs. Finally, it is

anticipated that additional studies would be required to parameterize the deposition of PAHs to land and aquatic surfaces and to assess deposition and reentrainment in indoor air.

Key Question 3: What are the relationships among sources of air toxics and aggregate human exposure and dose?

Individuals are typically exposed to POM of both outdoor and indoor origin while they are outdoors, commuting, and indoors. The different types of PAH sources result in concentrations of PAHs that vary among indoor environments and at different outdoor locations. The relative contributions of PAH concentrations in these different locations to total personal exposures depend significantly on the specific activities of different individuals and how much time they spend in each of the dominant source or exposure microenvironments. Refined exposure models for POM are needed to improve exposure assessments. The overall approach to developing a research program is to integrate models and measurements in an iterative process that would finally reduce urban, mobile, and indoor exposure uncertainty. A first-generation POM model would be developed and used as a screening level personal and population exposure and dose model. This initial model would be used to generate exposure and dose estimates for various cohorts and population groups of concern. The results would be used to identify gaps in knowledge and key sources of variability and uncertainty influencing the exposure and dose model predictions. Limitations that represent the greatest uncertainty or highest risk are identified as critical research needs. Laboratory and field measurement studies are conducted to address the highest priority needs. Models are then refined and evaluated based on the measurement data. Where needed, new methods are developed that would allow the appropriate data to be collected.

The following important data gaps have been identified that would be refined and prioritized based on the initial modeling work:

- Distribution of PAHs in outdoor microenvironments. [3B, near-term need] Information is needed on concentrations of PAHs in outdoor microenvironments in relationship to mobile sources and area sources, including wood burning and commercial charcoal grilling. This becomes important for urban areas where there are many potential sources and a large number of individuals who may be exposed. Understanding those geographical areas that are most highly impacted by area sources becomes an important issue for minority and low income

groups who may have disproportional risks because of where they live and for determining residual risk after implementation of MACT standards.

- Distribution of PAHs in selected indoor microenvironments. [3C, near-term need] Although data are available for homes, there are no or little data for other microenvironments, including automobiles, school buses, restaurants, and commercial buildings. Microenvironments for testing should be selected based on how common these microenvironments are, the proportion of time the general population or the population of concern spends in the microenvironment, variability in penetration factors or decay rates relative to residential setting, and anticipated indoor sources that have not been adequately characterized for PAH emissions.
- Relationship of personal exposure air concentrations to concentrations at ambient and indoor monitoring sites. [3C,E near-term need] Although data exist on indoor concentrations, personal exposure measurement data are generally not available. For nonsmokers, it is important to identify the highly exposed populations and to characterize their exposure. Understanding the relationship between personal exposure and indoor, outdoor, and ambient air concentrations is important to predict exposure. Data should be sufficient to describe the magnitude and variability, examine the inter- and intrapersonal variability, and identify and model the factors that contribute to variability in the relationships.
- Aggregated exposure to PAHs for all routes and pathways—especially for highly exposed or populations. [3D,E,H, long-term need] Data for young children suggest that dietary and nondietary ingestion are the predominate routes of exposure. Thus, it is important to conduct multipathway exposure monitoring. Urinary metabolites of the PAHs should be used to verify exposure estimates from multi-route sampling.
- Data to estimate lifetime exposures to PAHs. [3C,D,E,H, long-term need] The PAHs are carcinogens, thus risk assessments would be based on lifetime exposure estimates. Most of the currently available indoor air and personal exposure measurement data have been collected for very short periods (1 to 4 days). Information on variability in personal exposure over time should provide an opportunity to improve lifetime exposures. Urinary biomarkers may provide a relatively simple and inexpensive approach if sufficient data are available on sources and pathways.

Data needs can be met by conducting several well-designed field monitoring studies. Although details of studies are not provided, several fundamental concepts should be addressed. Studies, for the most part, should focus on urban environments, where there are

many potential sources, as well as many people who can be exposed. Studies should include low-income and minority populations who have opportunities for higher exposure, other potentially highly exposed groups, and susceptible subpopulations, such as children. All studies must collect ambient and indoor monitoring data so that relationships for estimating exposures as part of risk assessments can be developed quantitatively. Sufficient information must be collected to identify and quantify the factors associated with exposure variability.

- Refined exposure models. [3D,F, long-term need, but work must begin near term] Because the stochastic human-exposure-to-dose simulation-POM is a two-stage Monte-Carlo model that incorporates both variability and uncertainty in the model inputs and parameters, appropriate statistical distributions would be fit to the new field data gathered for this purpose. Variability in concentrations and exposure factors would be characterized by season and by proximity to major outdoor sources (such as traffic), housing factors, source emissions using mass-balance and regression based techniques. Uncertainty distributions would be fit to each of the key model parameters. The model for POM would be refined to incorporate these new distributions. Moreover, pertinent new exposure pathways that were not previously modeled or considered would be included. The model would be upgraded also to allow predictions of longer term exposures as longitudinal data become available. The initial model would include limited microenvironments (home, outdoor, in-car, etc.) but would be expanded to include nonresidential microenvironments, such as in schools, workplaces, restaurants, commuting by bus, etc.

Key Question 4: What are the effects of exposure to air toxics on human health?

The primary known adverse health effect from POM exposure is cancer (primarily lung, mammary gland, and skin cancer). The relative carcinogenic potencies of POM span a wide range from inactive to extremely potent. Exposure to POM occurs via multiple routes, and the composition of the mixtures to which individuals are exposed may change with exposure route. POM also has been associated with noncancer health effects, including low birth weight and other developmental effects, immunotoxicity, and cardiovascular disease, and also may exacerbate preexisting conditions such as asthma.

Much is known about the mode of action of many POMs, but many specifics remain to be elucidated. To exert carcinogenic effects, POM requires metabolic oxidation to form reactive intermediates that bind to cellular macromolecules to form adducts. The POM-deoxyribonucleic

acid (DNA) adducts have the potential to induce alterations in the DNA sequence that can induce the conversion of normal cells to cancer cells. The efficiency with which each of these steps occur may have a pronounced influence on the carcinogenic potency of a given POM. The efficiency of production of reactive metabolic intermediates is a product of the interactions of both the molecular structural features of individual POM compounds and the enzymes that perform the metabolic conversion. Multiple forms of the metabolizing enzymes exist, each with different degrees of expression in different tissues and at different developmental stages, and each with unique specificities for substrate and type of product produced. Evaluating the risk posed by individual POM requires understanding these underlying factors. [4D] Developing a clear understanding of the major enzymatic pathways for activation would provide an understanding of the influence of genetic variation in the metabolizing enzymes on the activation process. Similarly, cellular repair pathways that remove POM damage from DNA also may be significant modulators of risk, and genetic variability in components of these pathways may define additional sensitive subpopulations. Understanding these factors would provide a basis for identifying sensitive subpopulations and estimating the magnitude of altered susceptibility. [4F] This is an important area for long-term research.

The mechanism of action for POM carcinogenicity has suggested the use of stable POM-DNA adducts as useful biomarkers for the effective molecular dose of POM to cells in target tissues. New research has identified unstable POM-DNA adducts that may be related to dose and biological activity. Additional near- and long-term research is needed to understand the relationships between tissue dose of POM and the molecular dose to the DNA for both stable and unstable DNA adducts. [4G,H] Such biomarkers may serve an important role in helping to establish mechanistic concordance between animal and in vivo models and humans and among different exposure routes [4I], exposure regimens [4B], and dose ranges [4H].

The POMs may also act via additional pathways to induce cancer. New research suggests that some potent POM may influence the regulation of the p53 gene, leading to disruption of cell cycle checkpoints and altered cell cycle kinetics. This inhibits apoptotic mechanisms, allowing damaged cells to divide rather than die. This type of cellular disregulation increases the likelihood of carcinogenesis and may imply a higher risk than would be suggested by the level of DNA binding alone. Additional long-term research is needed to identify those POMs for which this mechanism is likely to be important and to determine the increased cancer risk conferred by this pathway. [4D]

Reactive POM metabolites also may form protein adducts, which may alter the normal biological functions of the cell and may induce a variety of secondary effects, including increased oxidative stress and lipid peroxidation, cytokine induction, and cytotoxicity, which may, in turn, promote carcinogenesis. [4C] This mode of action may be important in mediating inflammatory and immunotoxic responses. These adducts also may serve as useful markers of effective dose and biological effect. Additional near- to long-term research is needed to determine the health effects for which this mode of action is important. [4A]

The ubiquitous occurrence of POM as components of complex mixtures requires that EPA develop an understanding of how various POMs interact with each other and with other co-contaminants in inducing adverse health effects. The EPA's default assumption is that risks posed by a mixture of POMs is reflected by additivity of the risks posed by the individual constituents. Near- and long-term research also is needed to identify those cases where risk is significantly greater than that predicted by additivity. [4E]

An additional major area of uncertainty is the metabolism and biological activity of heterocyclic and substituted POM. Most of the available biological data are from studies of POM that contain only carbon and hydrogen, yet many environmental POM contain oxygen, sulfur, and nitrogen, substituents that greatly alter the chemical properties of the molecules and that greatly impact biological activity. These substituted POMs may be responsible for much of the carcinogenic potential of some POM mixtures, yet these represent a subclass with little experimental basis to support risk assessment. The SAR approaches, as well as lab studies, are needed to better identify the structural features of POM that may be important determinants of biological potency. Both near- and long-term research are needed to understand the contributions of POM substituents to risk. [4C,D]

Fewer mechanistic data are available on the mode of action of POM in inducing noncancer adverse health effects. One current area of concern is the potential for POM metabolites to bind to estrogen receptors and thyroid hormone receptors in the cell, disrupting normal hormonal regulation and function. Additional near-term research is needed to screen for endocrine disruption by specific POMs and POM metabolites and mixtures, as well as development of robust receptor-ligand binding models to predict the potential for untested POM and POM metabolites to disrupt endocrine function. [4A,C]

Further research also is needed to determine the modes of action for other noncancer adverse health effects. Hypotheses for the mode of action for the effects of POM on adverse

reproductive and developmental effects must be developed and tested in the near term, with particular emphasis on critical periods of exposure, tolerance, and increased sensitivity to POM effects. [4C,F] To assess the risk posed by POM to exacerbate asthma, airway markers suggesting the possibility of induction or exacerbation of asthma need to be evaluated in appropriate animal models in the near term. [4C] These markers could include the presence of cell damage or the production of an inflammatory response, certain cytokines, or nonspecific airway hyperreactivity. Such markers then would be applied to the identification of the specific POM components responsible for these effects in the long term. Following identification of the causative components, exposure-dose-response characterization is needed, with possible evaluation of the effects of multiple components on response. This step requires understanding the relative roles that concentration and duration of exposure might play.

Key Question 5: What are the hazards and dose-response relationships of air toxics?

Limited data are available on the health hazards of POM/PAHs, and even less on their quantitative dose-response relationships. Knowledge of these relationships will reduce uncertainty among the NATA. Additional cancer and noncancer dose-response assessments should be developed for POM. These dose-response assessments should include cancer unit risks, RfCs, and RfDs for chronic exposures. Currently, a toxicological review is being developed for benzo[*a*]pyrene. What is needed is a plan to develop toxicological reviews for PAHs, either individually or by using a toxic equivalency factor approach. All these dose-response assessments should be externally peer reviewed, EPA consensus reviewed, and then listed on IRIS for use in developing risk assessments. Although many of the PAHs are known to be very carcinogenic, sufficient data exist for very few that would permit development of toxicological reviews. Only a few cancer and no noncancer assessments for PAHs are present in the IRIS database. Inhalation and oral testing data to support dose-response assessments for POMs missing such critical data should be obtained through the use of EPA's test rule authority, and ORD should determine the exposure route and duration and toxicological endpoints for which testing should be conducted. [5A]

While acute noncancer dose-response assessment methods should be refined, and a framework developed for incorporating these values onto IRIS to provide better assessments of metal, aldehydes, halides, and hydrocarbons, determining the hazard from acute exposure to

POM/PAH is less of a priority based on their lower volatility and therefore minimally expected acute exposure by inhalation. [5B]

The noncancer dose-response assessment methods for chronic RfCs need updating. The current methodology for developing RfCs divides gases into three classes based on their reactivity and solubility and utilizes a paradigm that describes gas solubility using mass transfer coefficients. Gas models that can predict the dosimetry of reactive and nonreactive gases and water soluble and insoluble gases are needed. Thus, over the near term, mass transfer coefficients for POM are needed for the three categories of gases: Category 1, which are highly water soluble, rapidly reactive, and do not penetrate to blood; Category 2, which are water soluble and show accumulation in the blood; and Category 3, which are water insoluble and perfusion limited. Because POM often exists as particles, more precise models of the regional deposited dose ratio for particles of large and small mass median aerodynamic diameters also should be developed. The International Commission on Radiological Protection 66 model structure, with modifications of ventilation rate and activity patterns would be useful as a basis for developing such a dosimetry model for particles. Other longer term improvements needed for dose-response assessment of POM, as well as other air toxics, include additional approaches for modeling continuous data by the BMD method, harmonization of cancer and noncancer dosimetry, and route-to-route extrapolation methods. [5C]

Probabilistic dose-response methods also are needed over the longer term. Because dose-response assessments for cancer are based on the probability of an effect (tumor), cancer unit risks are derived from a probabilistic model. However, for noncancer endpoints, EPA has no definitive methodology for determining risk above reference levels such as the RfC. The basis of the RfC, which includes the application of either a single-point NOAEL or a statistical approach such as BMD (Crump et al., 1995), does not permit it to be used in probabilistic risk assessments, where knowing the probability of the occurrence of a health effect in a population is desired. Both the NOAEL and BMD approaches provide deterministic dose-response assessments, but not probabilistic ones. Categorical regression and Bayesian statistics may be useful in developing methods for probabilistic dose-response assessments for POM if large, adequate databases are available. Another approach that should be explored is the probabilistic characterization of the uncertainty about the human population threshold (i.e., the uncertainty factors used in current noncancer dose-response assessments are presented as probabilities in the

traditional RfC or RfD algorithm), but additional research is needed to determine the most feasible approaches. [5D]

Key Question 6: What are the risks posed to people exposed to air toxics?

To improve risk estimates among the NATA, better methods are needed to enhance uncertainty analysis. The result of poorly characterized variability or uncertainty of risk estimates is an imprecision that may give a systematic over- or underestimate of risk. Uncertainty analysis in risk assessments should include variability and uncertainty associated with individual and population cancer risk estimates. The analysis also should incorporate the relative contributions of individual models and input parameters to the overall uncertainty and variability in the risk estimate. Uncertainty analysis for risk assessment should be codified such that there is a systematic process for estimating individual sources of uncertainty and variability using a sensitivity approach. From such a process, adjustments of parameters such as activity patterns, microenvironmental exposures, or cancer unit risks could be weighted in their ability to affect risk expected from POM exposure under variable conditions or from POM compounds having different potency. Application of Monte Carlo analysis to estimate the simultaneous contribution of all the sources of uncertainty and variability also should be codified. With the current statistical knowledge, much of this codification can begin near term, whereas application examples can extend over the longer term. [6C]

A framework for incorporating mode-of-action data into risk characterization should be developed. Although the mode of action of some PAHs as a subset of POM is known, it is not known for many others. To develop cancer unit risk estimates under the new guidelines, it would be necessary to understand the binding and repair of exogenous agents to DNA, and how that affects receptor-mediated mechanisms of action and cancer induction and also mechanisms of toxicant interference with critical cellular pathways, such as signal transduction and receptors involved in cell growth. Gaps in the knowledge of mode of action as it relates to human susceptibility also present significant uncertainty in cancer and noncancer dose-response assessments. The National Research Council has recognized that, with respect to cancer, EPA does not account for person-to-person variations in susceptibility. Factors such as carcinogen metabolism, DNA-adduct formation, DNA-repair rate, synergistic effects of carcinogens, and age may contribute to different modes of action that influence susceptibility to cancer. As modes of action responsible for increased susceptibility of certain subpopulations (e.g., children,

elderly, asthmatics) are identified and described over the longer term they should be incorporated into risk assessment methods. [6A]

Although people are frequently exposed to a mixture of POM, risk characterization of such mixtures is not well developed. It is possible that the combined exposures to multiple POM compounds may produce either synergistic or antagonistic effects (i.e., effects either more detrimental or less detrimental than exposure to each pollutant individually). Recent epidemiological evidence indicates associations between air pollution and increased illness and death in humans is unlikely to be the result of exposure to a single compound. Research is needed on methods of extrapolation from toxicity information about one or more complex mixtures of POM (or other air toxics). Determination of comparative cancer potency for PAHs is one approach to gaining information about the toxicity of a mixture. Risk assessment approaches utilizing comparative potency to predict biochemical and toxicological responses other than cancer should be developed. Incorporating this type of mixture information into a risk assessment has not been done with sufficient rigor. Risk methods may include mathematical models for well-understood interactions, as well as decision frameworks for handling sparse and qualitative data. A key risk assessment that follows the proposed *Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (U.S. Environmental Protection Agency, 1999b) should be conducted over the near term as an example of how to conduct such risk assessments for POM as well as other air toxics mixtures. In light of assessment needs dictated by the CAA Amendments, there is a need to determine both chemical pairs for toxicologic interactions testing and complex mixtures for whole mixture toxicity testing over the near term. [6B]

The underlying biological basis of POM toxicity would be identified through health effects work proposed in this strategy. Biological markers (biomarkers) of toxicity thus would be elucidated. Biomarkers of susceptibility likely would emerge as research on health effects of POM progresses. Biological measures of exposure also are proposed as needed research in this strategy. Already, the National Human Exposure Assessment Survey (e.g., urine, blood, hair, and nails) would assist in describing exposure. Because a biomarker of effect or response is qualitatively or quantitatively predictive of health impairment or potential impairment, depending on the level of exposure, it has the potential to be useful in risk assessment. In the most optimistic sense, the use of biomarkers should permit the assessment of risk at lower levels of exposure than might be possible through the use of dose-response information only. Biomarkers of effect represent points on a continuum of disease, and the relationship of the

concentration or presence of a biomarker to risk needs to be established before certainty and specificity of biomarkers can be used in risk assessment. Risk assessment methods would need to be developed over the long term that can utilize biomarkers of effect or exposure to improve NATA activities. Stable POM-DNA adducts may be useful as biomarkers of molecular dose and, therefore, of cancer risk, but this relationship needs to be determined, and risk assessment methods developed to utilize such relationships. To assess the risk of lung inflammation (e.g., macrophages), cell changes (e.g., lactate dehydrogenase), or increased permeability of the alveolar-capillary barrier (e.g., serum protein) on exposure to POM, the relationship of bronchoalveolar lavage fluid results to risk of adverse effects needs to be determined. [6D]

Key Question 7: How can risks from air toxics be prevented and managed cost effectively?

Effective risk management involves the identification of risks, formulation of risk management objectives, assessment of risks, and application of regulatory or other strategies to prevent or reduce the risks from pollution to the environment. The goal of risk management research is to develop and assess methods and technologies for pollution control and prevention. It is the intention of the ATRS to control groups of air toxics. Relevant research is needed on both a near-term and long-term basis.

Many air toxics, including POM/PAH, are formed as the result of combustion processes. To better understand how these pollutants are formed in various industrial and combustion processes, near-and long-term research to examine the formation processes and destruction pathways of POM from stationary combustion devices would be performed to get a better understanding of how pollutant formation could be prevented. [7A,D]

To better understand how industrial pollution prevention (P2) activities affect emissions, the influence on emissions from combustion sources whose feedstocks are influenced by those P2 activities (e.g., the influence of recycling on emissions of POM from municipal waste combustors) would be examined. This is both a near- and long-term research need. [7C]

Near-term demonstrations of metal CEMs, in coordination with the U.S. Department of Energy, would be performed to show how CEMs for controlled streams would be beneficial for compliance determination and risk management purposes. [7E]

Cost-effective compliance monitors, capable of measuring multiple air toxics are needed for both near and long term. Research to develop the application of innovative CEMs, such as

Jet-REMPI and on-line gas chromatography for use as a system diagnostic tool, MACT screening tool, and research tool would be conducted. [7F]

Emissions control and P2 alternatives for area source categories are needed, mainly on a near-term basis. The identification of P2/control alternatives for POM emissions from application of FIFRA consumer products (in coordination with EPA's Office of Air Quality Planning and Standards consumer product rulemaking action), open burning sources (forest and prescribed burning and open burning of scrap tires), and gasoline distribution Stage I and II controls would be examined. For forest and prescribed burning, work would be conducted in cooperation with the U.S. Department of Agriculture's Forest Service. [7G]

Pollution prevention programs for indoor environments are needed. Following risk assessments for combustion processes in indoor nonresidential environments, research on potential engineering solutions for risk reduction/pollution prevention in indoor environments would be initiated, if warranted. The cost-effectiveness of these options and their relative ability to manage risks compared to outdoor sources/control options also would be evaluated. [7B,I,J,L]

No specific emissions control/P2 research needs for POM/PAH from mobile sources were identified. The POM/PAH emissions reductions from mobile sources are expected as a co-benefit of other mobile source emissions control programs. [7H]

4. IMPLEMENTATION AND OUTCOMES OF THE ATRS

4.1 INTRODUCTION

This chapter describes how ORD expects to implement the ATRS. Implementation first involves laying out the expected major accomplishments, and then undertaking activities as defined in an Air Toxics Multi-Year Plan (MYP) to achieve these accomplishments. As a key part of ATRS implementation and MYP, scientist-to-scientist meetings will provide input into the ORD's planning processes, in which priorities are set and specific activities are assigned.

4.2 IMPLEMENTATION OF THE AIR TOXICS RESEARCH STRATEGY

This section provides an overview of how the research strategy will be implemented in the context of ORD's planning processes. Key components of the ATRS implementation process are the development of a multi-year plan, appointment of a laboratory specific and cross laboratory steering committees, and the execution of periodic scientist-to-scientist meetings to identify the largest uncertainties and greatest programmatic needs to address within the plan using the most up to date science.

4.2.1 Developing and Updating a Multi-Year Plan

Research plans must be developed before ORD can make the most efficient use of its resources to provide results that will aid OAR in its NATA activities. The Air Toxics Multi-Year Plan (MYP) will be consistent with the ATRS and the individual ORD laboratory and center research strategies, plans, or steering committees as described below.

Through 2018, develop and improve (1) air quality models and source recepor tools to identifiy the sources and source contributions of hazardous air pollutants; (2) cost-effective pollutoin prevention and other control options to address indoor an urban pollutant sources that significantly contribute to risk; and (3)develop and improve scientific information and tools for quantitqative assessment of nationwide, urban, and residual air toxic risks to susceptible populations from hazardous air pollutants, considering both indoor and ambient air environments.

Figure 7. Air toxics subobjective

The initial MYP was developed using the expected major accomplishments of the ATRS (see Table 7). These major accomplishments support the ORD air toxics subobjective as seen in Figure 7, which supports the GPRA air goal (Figure 3). The MYP begins with an overarching statement of the long-term goals that covers the period from 2003 to approximately 2008. Annual Performance Goals (APGs) have been developed that specify how ORD plans to achieve the long-term goals. The APGs will be accomplished by producing various outcomes that will be listed in the MYP as Annual Performance Measures (APMs). The integration of the APMs within laboratories and centers to achieve APGs and the cross-laboratory and center integration of APGs will be described in an accompanying narrative of the MYP.

4.2.2 Steering Committees and Scientist-to-Scientist Meetings

Laboratory specific or cross laboratory steering committee(s) may be appointed with at least one representative from each of the participating ORD laboratories and centers, OAR, and Regions. The steering committees tasks will include planning, organizing, and holding scientist-to-scientist meetings and participating in the development of a multi-year research plan for ORD's laboratories and centers. As an advocate of air toxics research the primary role of the steering committee is to develop a laboratory research Implementation Plan, prioritize research, monitor progress, and communicate results. The National Health and Environmental Effects Laboratory has already initiated such an effort to address the challenging task of addressing health effects research needs given resource and staffing limitations of the program.

Scientist-to-scientist meetings proposed within the ATRS will provide ongoing scientific input into directions for the MYP. Future scientist-to-scientist meetings and steering committee efforts will make recommendations to the Air Research Coordination Team on how the MYP should be altered in keeping with emerging research results and needs.

The goals for scientist-to-scientist meetings will be to:

- engage principal investigators and other scientists in the air toxics issue and to put them in a position where they understand the issues and background information,
- gather information and perspective from scientists to help in designing and executing an ATRS-based research program on air toxics and including the results in ORD research plans,
- to foster cross-laboratory projects by increasing face-to-face contact between investigators in different labs

- keep investigators from the different labs, assistant laboratory and center directors, program office staff, and associate directors abreast of the latest science and programmatic issues;
- help individual scientists see where their work fits into the big picture; and
- foster and measure progress.

Both the steering committee meetings and the scientist-to-scientist meetings help to effectively communicate research findings to OAR (and others) and will generate dialog between the program office and investigators on how best to use the research findings in the regulatory arena. These meetings will provide a model for fostering cross-laboratory research and communication between the laboratories and OAR. Ultimately, these meetings also would provide the first indications that some tasks have been completed (a sure sign of progress) or require some redirection and that other tasks need to be initiated. Recommendations for future changes in strategic direction may arise from the steering committee meetings or the scientist-to-scientist meetings.

4.3 EXPECTED OUTCOMES

As ORD implements the ATRS, a series of major accomplishments will result. The main purpose of these accomplishments is to reduce the uncertainties associated with assessing and managing air toxic risks. To reduce the uncertainties in NATA, ORD can be expected to develop and improve (1) air quality models and source receptor tools to identify the sources and source contributions of air toxics; (2) scientific information and tools for quantitative assessment of nationwide, urban, and residual air toxics risk, including risk to susceptible populations from air toxics considering both indoor and air environments; and (3) cost-effective pollution prevention and other control options to address indoor and urban pollutant sources that significantly contribute to risk.

Table 10 lists the expected outcomes from this strategy. Initially the research will focus on the four HAP groups, but may expand to other HAP groups as appropriate. To do so, ORD will develop data to study and identify where grouped chemicals facilitate an increased understanding of dose-response and mode-of-action, exposure, risk characterization, and control of air toxics. The ORD will first test the preliminary chemical structure/reactivity classification approach that was used to group the list of air toxics. As the ATRS is implemented it is likely

TABLE 10. EXPECTED OUTCOMES

Initial Major Accomplishments - FY02 to FY05	Continuing Major Accomplishments - FY05 Onward
*Develop data to study and identify where grouped chemicals in the four air toxics groups facilitate increased understanding of dose- response and mode-or-action, exposure, risk characterization, and control of air toxics.	*Determine where proposed air toxics groups facilitate more rapid discovery of dose-response, exposure, and assessment, and control parameters.
*Reduce significant uncertainties in NATA as follows: • better describe emissions, fate, exposure-dose-response, and risk characterization to air toxics from urban, mobile, and indoor air sources; • better describe emissions, fate, exposure-dose-response, and risk characterization of air toxics pertinent to 4/7-year residual risk standards; and • incorporate research results into the second national-scale assessment of NATA.	*Adjust chemical groupings based on new research findings. *Continue to reduce uncertainties in NATA as follows: • with an emphasis on susceptible populations and mixtures better describe emissions, fate, exposure-dose-response, and risk characterization of air toxics from urban, mobile, and indoor air sources; • better describe emissions, fate, exposure-dose-response, and risk characterization of air toxics pertinent to 7/10-year residual risk standards; and • incorporate research results and modeling tools into the third (and beyond) national-scale assessment of NATA.

other HAP groups or subgroups will be identified and studied. The research also will be used to address uncertainties found during evaluation of the initial national-scale assessment and subsequent national-scale assessment, as well as other NATA activities.

The time line for completing these accomplishments, in part, depends on the various research disciplines and must account for the way science is conducted most effectively in each discipline. For research into emissions and exposure-related questions (Key Questions 1, 2, and 3), the general scientific approach is as follows:

- create a first-generation model for priority air toxics,
- identify sources and potentially susceptible populations for priority air toxics,
- identify key exposure data needs,
- conduct studies on aggregate and cumulative exposure data for general population and susceptible populations that address key exposure data needs,
- develop generic exposure data for the general population and susceptible populations (i.e., activity and location information, metabolic data), and
- use these data to refine and evaluate exposure models.

Exposure studies, generally are expensive and require several years to plan, conduct, and analyze the data. In addition, monitoring methods allow multiple chemicals to be monitored at one time.

Thus, it is anticipated that priority air toxics, susceptible populations, and mixtures could be addressed through a single study or several related studies.

Within health effects research (Key Questions 4 and 5), the general sequence of activities will be first to understand the effects of individual compounds, including the mode-of-action, relationships among exposure, dose to target tissues, and outcomes, and structure-activity relationships. The initial understanding of the important health outcomes and dose-response relationships will help generate hypotheses about likely susceptible populations. For example, if the lung is an important target site of toxicity at low dose levels, then people with asthma or other pulmonary diseases potentially would be susceptible populations. Mode of action information, typically developed after dose response evaluations, also can lead to development of hypotheses about likely susceptible populations, as well as suggest combinations of chemicals that might produce a greater than additive toxicity. Information about prevalent co-exposures also will help identify the most important mixtures to study. For this reason, the majority of research on mixtures will follow development of information about the individual chemicals, their modes of action, and likely co-exposures. Of course, the quality and quantity of information available for specific air toxics vary, and research sequences likely will vary accordingly, but, in general, the research will attempt to first understand hazards of individual compounds, followed by study of susceptible individuals and, finally, mixtures.

Risk characterization (Key Question 6) is a function of dose-response assessment and exposure assessment and describes the magnitude and uncertainty of risk. Therefore, research developments that improve the knowledge of dose-response and exposure will provide the basis for improving methods of risk characterization by better defining the magnitude and uncertainty of risk. Currently and in the near future, research results that improve the understanding of dose-response and population exposures to individual air toxics will be the basis to support risk assessment methods used in the early NATA and other assessment activities. As personal exposures and health effects of susceptible subgroups exposed to air toxics are better defined, improved risk characterization methods to take advantage of emerging results on susceptible populations will be developed to aid programmatic assessments. Understanding dose-response and personal exposures to single air toxics for both healthy and susceptible subgroups then will permit future research on mixtures. New risk characterization methods will improve NATA by characterizing the magnitude of risk from mixtures, while defining the risk characterization uncertainties.

In general, risk management research (Key Question 7) will address high-risk sources. The various NATA assessments will provide a basis for selecting the universe of high-risk sources by which to expand the risk management understanding and options. From this universe, difficult management issues will be addressed, tending to move from high-volume source (including indoor air) issues associated with susceptible populations to ubiquitous sources associated with mixtures.

REFERENCES

Crump, K. S.; Allen, B. C.; Faustman, E. M. (1995) The use of the benchmark dose approach in health risk assessment. Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum; report no. EPA/630/R-94/007. Available from: NTIS, Springfield, VA; PB95-213765/XAB.

EH&E. 2000. Ranking Air Toxics Indoors. Draft Report. Prepared for the Indoor Environments Division, U.S. Environmental Protection Agency, by Environmental Health and Engineering, Inc., Newton, MA. EH&E Report #11863.

Federal Register. (1999) National air toxics program: the integrated urban strategy; notice. F. R. (July 19) 64: 38,705-38,740. Available: www.epa.gov/ttn/uatw/urban/urbanpg.html [2000, July 19].

Keith, L. H.; Walker, M. (1995) Handbook of air toxics: sampling and analysis and properties. Boca Raton, FL: CRC Press.

U.S. Environmental Protection Agency. (1993) Motor vehicle-related air toxics study. Ann Arbor, MI: Office of Mobile Sources; report no. EPA/420/R-93/005. Available from: NTIS, Springfield, VA; PB93-182590/XAB.

U.S. Environmental Protection Agency. (1997a) EPA strategic plan. Washington, DC: Office of the Chief Financial Officer; report no. EPA/190-R-97-002. Available: www.epa.gov/ocfopage/plan/plan.htm.

U.S. Environmental Protection Agency. (1997b) Mercury study report to Congress [volumes I-VIII]. Washington, DC: Office of Air Quality Planning and Standards, Office of Research and Development; report no. EPA-452/R-97-003. Available: www.epa.gov/oar/mercury.html [2000, June 19].

U.S. Environmental Protection Agency. (1998a) National air quality and emissions trends report, 1997. Research Triangle Park, NC: Office of Air Quality Planning and Standards; report no. EPA 454/R-98-016. Available: www.epa.gov/oar/aqtrnd97/ [2000, April 14].

U.S. Environmental Protection Agency. (1998b) Toxics release inventory. Washington, DC: Office of Information Analysis and Access. Available: www.epa.gov/tri/index.htm.

U.S. Environmental Protection Agency. (1998c) Methods for exposure-response analysis for acute inhalation exposure to chemicals: development of the acute reference exposure. External review draft. Research Triangle Park, NC: Office of Research and Development, National Center for Environmental Assessment; report no. EPA/600/R-98/051.

U.S. Environmental Protection Agency. (1998d) Utility air toxics report to Congress.

U.S. Environmental Protection Agency. (1998e) Reports to Congress on the Great Waters.

U.S. Environmental Protection Agency. (1999a) Estimation of motor vehicle toxic emissions and exposure in selected urban areas. Volume I. Volume II: detailed toxics emissions and exposure estimates [draft]. Ann Arbor, MI: Office of Mobile Sources; report no. EPA-420-D-99-002a-b.

U.S. Environmental Protection Agency. (1999b) Guidance for conducting health risk assessment of chemical mixtures [external review draft]. Washington, DC: Office of Research and Development; report no. NCEA-C-0148.

U.S. Environmental Protection Agency. (2000a) Concept paper on air toxics monitoring [under review].

U.S. Environmental Protection Agency (2000b), National Air Toxics Program: The Integrated Urban Strategy Report to Congress, EPA-453/R-99-007.

U.S. Environmental Protection Agency (2001), Control of Emissions of Hazardous Air Pollutants from Mobile Sources, Fed. Regist. 66, pp. 17230-17273, March 29, 2001.

APPENDIX A

Overview of EPA's Air Toxics Program

The Four Components of EPA's Air Toxics Program

During the first 25 years of EPA's air toxics program, EPA regulated only a handful of HAPs, and in the 1980s, EPA supported state and local agencies to assist them in developing their own air toxic's programs. With the passage of the CAA Amendments of 1990, a shift in the approach to dealing with air toxics occurred. The overall approach to reducing air toxics under the CAA is to develop technology-based standards, and, subsequently, to implement a risk-based program to ensure the protection of public health and the environment. To respond to this mandate, the EPA structured an air toxics program containing four components (Figure A-1). This program is designed to characterize, prioritize, and equitably address the serious impacts of HAPs on the public health and the environment through a strategic combination of regulatory actions and studies, voluntary partnerships and initiatives, education and outreach, and ongoing research and assessments.

1. Source-Specific and Sector-Based Standards

Since 1990, EPA has made considerable progress in reducing emissions of air toxics through regulatory, voluntary, and other programs. To date, the air toxics program has focused on reducing emissions of toxic air pollutants from stationary sources through the implementation of technology-based emission standards. These emission standards are known as MACT standards and generally available control technology standards. Under this program, EPA listed for regulation 174 source categories that emit one or more of the 188 listed HAPs. As of January 9, 2001, EPA has promulgated 47 standards regulating 83 source categories. The EPA is continuing to develop these emission standards to cover the remaining source categories.

Further, the CAA contains provisions that have a risk-based focus. Section 112(f) of the CAA is designed to require EPA to assess the risk from source categories after MACT standards are implemented and for EPA to set additional standards if the level of residual risk does not provide an "ample margin of safety to protect public health" or "to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect."

(1) Source-Specific/Sector-Based Standards

MACT Standards
Residual Risk Standards
Urban Area Source Standards
Mobile Source Standards
Combustion Standards
Utility Determination and Actions

(2) National, Regional, Community-Based and Initiatives

Community-Based Assessment and Control Projects
Air/Water Interface Activities (including Great Waters Program)
Persistent Bioaccumulative Toxics Initiatives
Mercury Initiatives
Indoor Air Toxics Strategy
Structure to Integrated Federal and S/L/T Programs

(3) National Air Toxics Assessment Activities

Inventories
Ambient and Deposition Monitoring
Dispersion and Exposure Modeling
Risk Characterization

(4) Education and Outreach

Urban Air Toxics Reports to Congress
Air/Water Interface Program Outreach (including Great Waters)
Website Activities

Figure A-1. The four components of EPA's air toxics program.

The EPA also has issued final rules that establish new source performance standards for new solid waste combustion facilities and emission guidelines for existing solid waste combustion facilities. These rules set limits on emissions of mercury, as well as on those of dioxins and furans; from municipal waste combustors; hospital, medical, and infectious waste incinerators; and commercial and industrial solid waste incinerators. By the time these rules are fully implemented, they will reduce mercury and dioxin/furan emissions from these sources by about 90% and from current levels by more than 95%. The EPA is also working on rules to address other solid waste incinerators.

With respect to area sources, under the Integrated Urban Air Toxics Strategy (Strategy), EPA must ensure that 90 percent of the area source emissions of the 30 "area source" urban air toxics listed in the Strategy are regulated. In order to accomplish this, EPA identified 13 new categories of smaller commercial and industrial operations, or area sources, for regulation. The EPA plans to finalize regulations for these area source categories by 2004. The EPA has completed or nearly completed regulations on an additional 16 area source categories. However,

the Agency will be adding source categories, as needed, to the list for regulation to meet the requirement to regulate 90 percent of the area source emissions.

With respect to mobile sources, the EPA started enforcing the first federal emission standards for passenger cars in 1968. Since then, the Agency has developed emission standards for all types of highway vehicles, their fuels, and the engines used in virtually all varieties of mobile or portable nonroad equipment such as tractors, construction vehicles, recreational and commercial vessels, and lawn and garden equipment. In December of 1999, EPA finalized stringent new standards for all cars, light-duty trucks, and the gasoline they use.

In July 2000, EPA issued a final rule as part of the first phase of its two-part strategy to significantly reduce harmful diesel emissions from heavy-duty trucks and buses. The final rule is designed to significantly reduce harmful diesel emissions from heavy-duty trucks and buses beginning in 2004. In addition, under this rule, heavy-duty gasoline engines will be required to meet new, more stringent standards starting no later than the 2005 model year.

As part of the second phase of the strategy, in December 2000, EPA issued another final rule establishing a comprehensive national control program that will regulate the heavy-duty vehicle and its fuel as a single system. As part of this program, new emission standards will begin to take effect in model year 2007 and will apply to heavy-duty highway engines and vehicles. These standards are based on the use of high-efficiency catalytic exhaust emission control devices or comparably effective advanced technologies. Because these devices are damaged by sulfur, the rule also requires reductions in the level of sulfur in highway diesel fuel by 97 percent by mid-2006.

Although the toxics reductions from EPA's mobile source emission standards have been large, prior to 1990, EPA had no specific directions from Congress for a planned program to control toxic emissions from mobile sources. However, in 1990 Congress amended the Clean Air Act, adding a formal requirement to consider motor vehicle air toxics controls. Section 202(l) requires the Agency to complete a study of motor vehicle-related air toxics and to promulgate requirements for the control of air toxics from motor vehicles. EPA completed the required study in 1993 and has conducted analyses to update emissions and exposure analyses done for that study. In December 2000, EPA issued a final rule identifying 21 mobile source air toxics and setting new gasoline toxic emission performance standards. It also sets out a Technical Analysis Plan to continue research and analysis on mobile source air toxics. Based on the results of that research, EPA will conduct a future rulemaking, to be completed no later than

July 1, 2004, in which EPA will revisit the feasibility and need for additional controls for nonroad and highway engines and vehicles and their fuels.

Along with implementing the CAA, EPA has been providing information about air toxics in indoor air and how to mitigate exposures to these compounds. The indoor environment has particular relevance because people spend as much as 80 to 90% of their time indoors. Additionally, outdoor air is brought indoors through infiltration and mechanical ventilation, and there are also many sources of air toxics indoors. As EPA continues to develop and enhance knowledge of exposures and risks from indoor air toxics through the indoor environments program, EPA will seek to include information on indoor exposures in characterization of risk associated with outdoor sources and in the development of risk management options for air toxics.

Finally, the EPA has gathered data on the mercury emissions from coal-fired electric utility power generation plants to evaluate the need for regulation of air toxics from these sources. Utility plants (primarily coal-fired plants) emit approximately 50 tons per year of mercury nationwide, which is also one-third of the manmade mercury emissions in the United States. Mercury compounds are one of the listed 188 HAPs. There are a concern because mercury persists in the environment and can accumulate (e.g., can bioaccumulate in the food chain and lead to human exposure through food consumption). In December 2000, to reduce the risk mercury poses to health, EPA announced that it will regulate emissions of mercury and other air toxics from coal- and oil-fired electric utility steam generating units (power plants).

2. National, Regional, and Community-Based Initiatives to Focus on Multimedia and Cumulative Risks

The CAA requires a number of risk studies to better characterize risk to the public and the environment from air toxics. In response to this mandate, EPA has published the *Integrated Urban Strategy* (Federal Register, 1999), the *Urban Air Toxics Report to Congress* (U.S. Environmental Protection Agency, 2000), the *Utility Air Toxics Report to Congress* (U.S. Environmental Protection Agency, 1998d), the *Mercury Study Report to Congress* (U.S. Environmental Protection Agency, 1997b), and three *Reports to Congress on Deposition of Air Pollutants to the Great Waters* (U.S. Environmental Protection Agency, 1994, 1997 and 2000). These publications address specific issues identified in the CAA. For example, the *Integrated*

Urban Strategy has three goals for urban areas nationwide: (1) to ensure a 75% reduction in cancer incidence from stationary sources; (2) to ensure a "substantial" reduction in health risks from area sources; and (3) to ensure that disproportionate risks are addressed first, thus focusing efforts on sensitive populations or where there are geographic hot spots.

The EPA plans to conduct urban-scale assessments for a number of selected cities to serve as case studies that may be particularly useful as guidance for State, local, and Tribal program assessments. The first pilot is being conducted in Cleveland and considers a combination of stationary, mobile, and indoor air sources. EPA will also make available technical support and risk assessment tools as needed, for authorities that wish to conduct their own local assessments to analyze area-specific progress and intraurban disparities. The experience EPA will gain through these analyses will also help EPA refine future assessments.

Through these assessment efforts, EPA will encourage and support area-wide strategies developed by State, local, or Tribal air pollution control agencies. These risk initiatives will include State, local, and Tribal program activities consistent with the *Integrated Urban Strategy* on the local level, as well as federal and regional activities associated with the multimedia aspects of HAPs, such as the Great Waters program and initiatives concerning mercury, and other persistent bioaccumulative toxics (PBTs).

Another EPA initiative consists of collaboration between the air and water programs on the impact of air deposition on water quality (e.g., by accounting for the contribution of air deposition to the total maximum daily load of pollutants to a water body). This is described in an Air/Water Interface Workplan dated January 18, 2001. In addition, offices within EPA's air program are working together to assess the risks from exposures to air toxics indoors and to develop nonregulatory, voluntary programs to address those risks.

In January 2000 the EPA created the Integrated Air Toxics State/Local/Tribal Program Structure Workgroup, which met from April through August 2000. EPA created the workgroup to obtain advice on how to structure a program encompassing Federal, State, local, and Tribal authorities to collectively address air toxics risk. EPA created the workgroup under the Clean Air Act Advisory Committee, which EPA chartered in 1990 through the Federal Advisory Committee Act. To address the charge provided by EPA, the workgroup developed a report that contains a structure for a program to address the air toxics risk. The EPA plans to issue guidance and rulemaking to develop this program in the 2002 to 2003 time frame.

3. National Air Toxics Assessment Activities

EPA's NATA activities include all assessments conducted as part of EPA's air toxics program. Each assessment will be tailored for its intended purpose (e.g., measuring progress toward national risk reduction goals or assessing residual risks). As a result, assessments conducted as part of NATA will vary in scope and level of refinement. For example, NATA activities will encompass a variety of geographic scales (i.e., national, regional, urban/local). The NATA activities will help EPA identify risks, prioritize other air toxics program efforts, and track progress toward risk reduction goals and objectives. Figure A-2 shows how the NATA activities fit into the overall air toxics program, not only as one of the four components of the program, but also as the primary mechanism for informing and prioritizing efforts under the other three components and tracking progress towards the overall air toxics program goals.

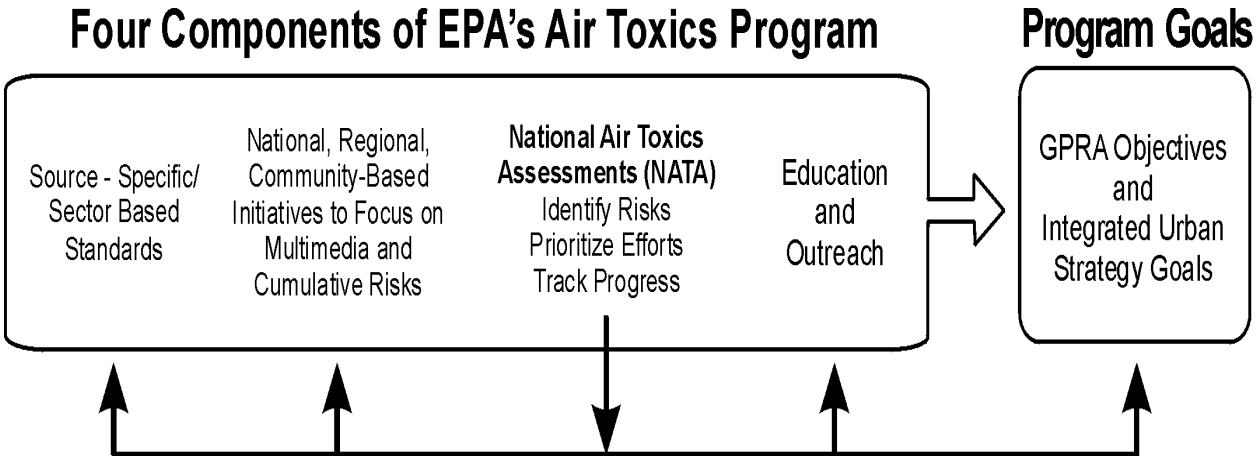

Figure A-2. The role of NATA activities in EPA's air toxics program.

The NATA activities include expanding air toxics ambient and deposition monitoring; improving and periodically updating emissions inventories; national- and local-scale air quality, multi-media, and exposure modeling (including modeling that considers stationary and mobile sources); and improving and using exposure and assessment tools. These activities will provide EPA and others with improved characterizations of air toxics risk for both stationary and mobile source programs.

Ambient air toxics information is a key component in supporting assessment activities, helping to characterize dispersion of HAP emissions and evaluating models and other assessment tools. Because of the importance of this information, EPA currently is working with State, local, and Tribal agencies to develop an approach to monitoring air toxics across the nation (U.S. Environmental Protection Agency, 2000). The primary purpose of this monitoring initiative is to characterize air toxics in the ambient air and to help understand the modeling tools used in NATA activities. The monitoring initiative calls for an initial pilot study network, which will yield results crucial to developing a National Network Design as well as provide local data for State, local, and Tribal agencies to use in assessing their own monitoring needs. The EPA also is compiling data from the many State, local, and Tribal toxics monitoring networks, and will analyze this data along with the pilot results during FY 2001 and FY 2002.

Over the past several years, EPA worked to build a national inventory of air toxics emissions. The EPA, with the assistance of State, local, and Tribal agencies, now has data sets for the 1990 to 1993 period and for 1996. The 1996 National Toxics Inventory (NTI) includes information generated from MACT standards development, as well as information provided by other regulatory authorities and various industries. The EPA is currently compiling the 1999 NTI, which will be available for modeling in June 2002.

The NATA activities include national and local-scale air quality and exposure assessments. For example, at the national level, EPA must assess the risk remaining (i.e., residual risk) after promulgation of technology-based standards (e.g., MACT standards) applicable to stationary sources. In general, EPA will base decisions on exposures predicted from modeling HAP emissions in air and, where appropriate, other media. Where available, EPA will include monitoring data as part of its analysis for refined assessments. The EPA will estimate the size and characteristics of the exposed population, and conduct uncertainty and variability analysis where appropriate. In analyzing residual risk, EPA will conduct risk assessments consistent with its human health and ecosystem risk assessment technical guidance and policies.

For NATA activities to be more complete, EPA will need not only to conduct multiple HAP assessments as in residual risk or multiple source assessments as in the urban program, but also should have adequate information and methods to conduct integrated and multimedia assessments. In the near future, EPA expects to use the Total Risk Integrated Model to address local- or neighborhood-scale applications. The CMAQ modeling system will support assessments on the urban-to-regional scale (and ultimately neighborhood scales) and will include

criteria pollutants as well as air toxics as EPA expands aggregate and cumulative risk assessment capabilities.

4. What Education and Outreach Do EPA Expect for Air Toxics?

The EPA believes that public participation is vital for the implementation of the overall air toxics program. The Agency is committed to working with cities, communities, State, local, and Tribal agencies, and other groups and organizations that can help implement activities to reduce air toxics emissions. For example, the Agency expects to work with the cities and other interested stakeholders in the national air toxics assessments that will be conducted. In addition, EPA will continue to work with stakeholders on regulation development. The Agency intends to work with local communities (including multiple stakeholder groups) in the development of local risk initiatives such as the urban community-based pilot projects.

The EPA published the first Urban Air Toxics Report to Congress in September of 2000. The EPA is required under the Act to provide two reports to Congress on actions taken to reduce the risks to public health posed by the release of toxic air pollutants from area sources. The Act also requires that the reports identify specific metropolitan areas that continue to experience high risks to public health as a result of emissions from area sources. The first report provides specific information about the Integrated Urban Air Toxics Strategy, including further details on the methodologies EPA used to develop the final urban air toxics list and the list of source categories. The second report is due in 2004. EPA also expects to report to the public about air toxics emissions trends and air quality in urban and other areas in its annual Air Quality and Emissions Trends Report in the future.

The Act directs EPA to monitor, assess and report on the deposition of toxic air pollutants to the "Great Waters," which include the Chesapeake Bay, Lake Champlain, the Great Lakes, National Estuary Program areas, and National Estuarine Research Reserves. Activities include assessing deposition to these waters by establishing a deposition monitoring network, investigating the sources of pollution, improving monitoring methods, evaluating adverse effects, and sampling for the pollutants in aquatic plants and wildlife. Pollutants of concern to the Great Waters include mercury, lead, cadmium, nitrogen compounds, polycyclic organic matter/polynuclear aromatic hydrocarbons (POM/PAHs), dioxins and furans, PCBs and seven banned or restricted pesticides. As part of the Great Waters Program, EPA funded special

monitoring studies at eight different coastal areas. In addition, EPA is expanding the National Atmospheric Deposition Program to include more coastal sites for long-term deposition records. EPA will continue to develop coastal monitoring and to support improvement of air deposition monitoring methods.

The Great Waters program is multimedia in nature and requires cross-program approaches to investigate and address problems. EPA's air and water programs are working together on two pilot studies to address mercury deposition to waterways, and the outcome of this effort will influence the development of joint national guidance for addressing Total Maximum Daily Loads (TMDLs) where air deposition is a factor. TMDLs specify the amount of pollutant that may be present in the water and still allow the water body to meet State water quality standards. TMDLs allocate pollutant loads among pollution sources (e.g., point and nonpoint sources), and include a margin of safety that accounts for uncertainty in the relationship between pollutant loads and characteristics of the waterbody. In part because of the efforts of the Great Waters program, there is now a greater level of coordination among research agencies and institutions to target areas of critical uncertainty and suspected threats to human health and the environment. Recent research continues to show that the diffuse emissions of urban areas can significantly affect nearby deposition rates to water bodies. The EPA recently completed an Air Water Interface Workplan, which details measures to protect both public health and our nation's waterbodies from atmospheric deposition of pollutants. This plan will be revised and reissued every two years.

Also the National Air Toxics Program/Integrated Urban Air Toxics Strategy recognized that, although exposures to air toxics indoors may be significant, the risks associated with indoor exposures are not as well characterized as those for exposures outdoors. In the July 19, 1999 Integrated Urban Air Toxics Strategy Notice, EPA stated that it would assess the current information on exposures to indoor air toxics, include information on indoor exposures in characterization of risks and in the development of risk management options for air toxics, and conduct additional research on indoor exposures to air toxics. The Indoor Air Toxics Strategy, a plan under development at EPA to reduce risks from toxic air pollutants indoors, will detail our initial approach to address those needs. It will provide an evaluation of past and current information on the potential exposures to, and risks from, air toxics indoors, and it will briefly describe the overall National Air Toxics Program and how indoor air toxics fits within the program. It will also present actions that have been taken in the past to reduce the risks from air

toxics indoors. The Indoor Air Toxics Strategy will also present a screening-level ranking and selection of key air toxics indoors, which was performed to help EPA prioritize its future efforts in this area. Finally, it will present the next steps in EPA's strategic approach to addressing indoor air toxics as a part of the National Air Toxics Program, building upon the current information and relying heavily on voluntary, nonregulatory efforts to reduce risks from air toxics indoors. An SAB review of the ranking analysis is planned for spring of 2001. After the SAB review, EPA will respond to any comments on the ranking analysis. The analysis will be finalized when the Indoor Air Toxics Strategy is released in late 2001.

The EPA will continue to develop and maintain Web sites with information on the urban air toxics program, the National Air Toxics Assessment and other air toxics programs. This includes coordination with State, local, and Tribal agencies on the presentation of results for the National-Scale Air Toxics Assessment.

APPENDIX B

Organic Chemistry Classification of HAPs, Their Relevance to Air Programs, and Availability of Physical Constants and Risk Information

This appendix presents the list of HAPs with information considered in developing the ATRS. Table B-1 is organized by HAP groups according, in general, to established chemical classification schemes used in organic chemistry. In addition to physical/chemical and structural/reactivity families, other aspects to the approach includes mechanistic families of chemicals (alkylating/arylating/acylating reagents, etc.). The resulting chemical classification led to 11 groups/classifications including the (1) alcohols/glycols; (2) aldehydes/ketones/other potential acylating agents; (3) alkylators (epoxides, aziridines, and others); (4) amines/precursors/derivatives; (5) ethers/alkyl and aryl; (6) halides/alkyl and aryl; (7) hydrocarbons/aromatic and nonaromatic; (8) metals/minerals and other inorganics; (9) organic acids/precursors/derivatives; (10) phenols and derivatives; and (11) sulfur/phosphorus-containing compounds. In this scheme, some chemicals can appear in more than one group when they have multiple chemical functionality and associated properties.

For each HAP, Table B-1 identifies the relevance of each HAP to the air toxics program objectives. That is, the table indicates (with a "Y") whether the HAP was listed as an urban air toxics pollutant, whether the HAP is associated with mobile source, indoor source, Great Waters, or the PBT program. The number of MACT standards addressing individual HAPs is presented as a measure of the potential for residual risk assessments.

Table B-1 contains qualitative information about the properties of the HAPs, especially in the context of exposure assessments. These properties are aimed at the HAP's physicochemical persistence (half-lives in environmental compartments) and potential bioavailability in the environment. Lipophilicity (e.g., K_{ow}) indicates solubility of a HAP in fat or the potential for biological uptake of a HAP. It is indicated as either lipophilic (Y) or not lipophilic (N). Henry's Law (fugacity) indicates the potential for a compound to move to the air media from another media, phase distribution, high (H), medium (M), or low (L). Sorption (K_s or K_{oc}) indicates the potential for a HAP to be stored in an organism after exposure to the HAP, bioavailable (Bio) or

TABLE B-1. AIR TOXICS

			Program Factors						Physical Properties						Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Polycyclic Organic Matter (POM)	Polycyclic Organic Matter (POM)		Y	Y		Y	Y	13	Y	L	Bio	Y					
Alcohols/Glycols	Diethanolamine	111-42-2															
	Ethylene Glycol	107-21-1						21								Y	100
	Methanol	67-56-1						34								Y	1,000
Aldehydes and Ketones/Acylating Agents	Acetaldehyde	75-07-0	Y	Y	Y			19	M	H	Bio	Y	Y	Y	1,000		
	Acetophenone	98-86-2														Y	3,000
	Acrolein	107-02-8	Y	Y					M	H	Bio			Y	1,000		
	Butanone, 2-	78-93-3						29									
	Chloroacetophenone	532-27-4												Y	1,000		
	Formaldehyde	50-00-0	Y	Y	Y			39	Y	H	Bio	Y	Y			Y	100
	Hydroquinone	123-31-9							N	L	Bio	Y					
	Isophorone	78-59-1														Y	1,000
	Methyl-2-Pentanone, 4-	108-10-1															
	Propionaldehyde	123-38-6															
	Quinone	106-51-4															

TABLE B-1 (cont'd). AIR TOXICS

			Program Factors						Physical Properties					Risk Values				
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Alkylators	Diethyl Sulphate	64-67-5															
	Dimethyl Sulphate	77-78-1															
	Epichlorohydrin	106-89-8											Y	Y	300		
	Epoxybutane, 1,2-	106-88-7												Y	300		
	Ethylene Oxide	75-21-8	Y					2									
	Ethyleneimine	151-56-4															
	Propiolactone, B-	57-57-8															
	Propylene Oxide	75-56-9											Y	Y	100		
	Propylenimine, 1,2-	75-55-8															
	Styrene Oxide	96-09-3															
Amines	Acetylaminofluorene, 2-	53-96-3															
	Aminobiphenyl, 4-	92-67-1															
	Aniline	62-53-3							Y	M	Bio	Y		Y	3,000		
	Anisidine, O-	90-04-0															
	Benzidine	92-87-5											Y			Y	1,000
	Diethylaniline, N,N'-	91-66-7															
	Dimethoxybenzidine, 3,3'-	119-90-4															
	Dimethylaminoazobenzene	60-11-7															

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties						Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Amines (cont'd)	Dimethylbenzidine, 3,3'-	119-93-7															
	Dimethylhydrazine, 1,1-	57-14-7															
	Dinitro-2-Methylphenol, 4,6-	534-52-1															
	Dinitrophenol, 2,4-	51-28-5														Y	1,000
	Dinitropyrene, 1,8-	42397-65-9					Y		Y	L	Bio	Y					
	Dinitropyrene, 1,6-	42397-64-8					Y		Y	L	Bio	Y					
	Dinitropyrene, 1,3-	75321-20-9					Y		Y	L	Bio	Y					
	Dinitrotoluene, 2,4-	121-14-2							Y	L	Bio	Y				Y	100
	Diphenylhydrazine, 1,2-	122-66-7											Y				
	Hydrazine	302-01-2	Y						Y	H		Y	Y				
	Methyl-1-Nitrosourea, 1-	684-93-5															
	Methylenedianiline, 4,4'-	101-77-9															
	Methylhydrazine	60-34-4															
	MOCA	101-14-4															
	Nitrobenzene	98-95-3							Y	H	Bio	N	D			Y	10,000
	Nitrobenzo[*a*]pyrene, 6-	63041-90-7															
	Nitrobiphenyl, 4-	92-93-3															

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Amines (cont'd)	Nitronaphthalene, 1-	86-57-7															
	Nitronaphthalene, 2-	581-89-5															
	Nitroperylene, 3-	20589-63-3															
	Nitrophenol, 4-	100-02-7															
	Nitropropane, 2-	79-46-9												Y	1,000		
	Nitropyrene, 1-	5522-43-0															
	Nitrosodimethylamine, –	62-75-9											Y				
	Nitrosomorpholine	59-89-2															
	Parathion	56-38-2															
	Quinoline	91-22-5	Y														
	Quintozene	82-68-8															
	Toluenediamine, 2,4-	95-80-7											D	Y	300	Y	1,000
	Toluidine, o-	95-53-4															
	Triethylamine	121-44-8							N	M	L	Y				Y	1,000
	Trifluralin	1582-09-8												Y	3,000		

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Ethers	Chloromethyl Methyl Ether	107-30-2															
	Dibenzofuran	132-64-9					Y		Y	L	Bio	Y					
	Dioxane, 1,4-	123-91-1							Y	M	Bio	N					
	Methyl Tert-Butyl Ether	1634-04-4							N	H	Bio	N		Y	100		
Halides	2,4-D	94-75-7														Y	100
	Allyl Chloride	107-05-1												Y	3,000		
	Benzotrichloride	98-07-7															
	Benzyl Chloride	100-44-7															
	Bis(2-Chloroethyl) Ether	111-44-4											Y				
	Bischloromethyl Ether	542-88-1											Y				
	Bromoform	75-25-2											Y			Y	1,000
	Carbon Tetrachloride	56-23-5	Y					12	Y	H	Bio	N	Y			Y	1,000
	Chlordane	57-74-9				Y	Y						Y	Y	1,000	Y	300
	Chloroacetic Acid	79-11-8															
	Chlorobenzene	108-90-7						11								Y	1,000
	Chlorobenzilate	510-15-6														Y	300
	Chloroethane	75-00-3												Y	300		
	Chloroform	67-66-3	Y		Y			13					Y			Y	1,000

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Halides (cont'd)	Chloroprene	126-99-8															
	DDE, P,P'-	72-55-9					Y		Y	L	Bio	Y					
	Dibromochloropropane	96-12-8												Y	1,000		
	Dichlorobenzene, P-	106-46-7					Y		Y	L	Bio	Y		Y	100		
	Dichlorobenzidine, 3,3'-	91-94-1					Y										
	Dichloroethane, 1,1-	75-34-3							Y	H	Bio	Y					
	Dichloroethane, 1,2-	107-06-2							Y	H	Bio	Y	Y				
	Dichloromethane	75-09-2	Y		Y			26					Y			Y	100
	Dichloropropane, 1,2-	78-87-5												Y	300		
	Dichloropropene, 1,3-	542-75-6	Y											Y	30	Y	10,000
	Dichlorvos	62-73-7												Y	100	Y	100
	Ethylene Dibromide	106-93-4							Y	H	Bio	Y	Y				
	Heptachlor	76-44-8					Y		Y	L	Bio	Y	Y			Y	300
	Hexachlorobenzene	118-74-1	Y			Y	Y		Y	L	Bio	Y	Y			Y	100
	Hexachlorobutadiene	87-68-3					Y		Y	L	Bio		Y				
	Hexachlorocyclopentadiene	77-47-4														Y	1,000
	Hexachloroethane	67-72-1							Y	M	Bio	N	Y			Y	1,000
	Lindane	58-89-9				Y			Y	L	Bio	Y				Y	1,000

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values				
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Halides (cont'd)	Methyl Chloride	74-87-3						15									
	Methyl Bromide	74-83-9							N	H	Bio	Y		Y	100		
	Methyl Iodide	74-88-4															
	Pentachlorophenol	87-86-5					Y		Y	L	Bio	Y				Y	100
	Polychlorinated Biphenyls	1336-36-3	Y			Y	Y		Y	L	Bio	Y	Y				
	TCDD, TCDF		Y	Y			Y		Y	L	Bio	Y					
	Tetrachloroethane, 1,1,2,2-	79-34-5	Y										Y				
	Tetrachloroethylene (PERC)	127-18-4	Y		Y			14								Y	1,000
	Toxaphene	8001-35-2				Y	Y		Y	L	Bio	Stable					
	Trichlorobenzene, 1,2,4-	120-82-1											Y				
	Trichloroethane, 1,1,1-	71-55-6						14	Y	H	Bio	N	D			Y	1,000
	Trichloroethane, 1,1,2-	79-00-5							Y	H	Bio	N					
	Trichloroethylene	79-01-6	Y		Y			17	Y	H	Bio	N	Y			Y	1,000
	Trichlorophenol, 2,4,6-	88-06-2							Y	M	Bio						
	Trichlorophenol, 2,4,5-	95-95-4							Y	M	Bio		Y				
	Vinyl Chloride	75-01-4	Y						Y	H	Bio	Y					
	Vinyl Bromide	593-60-2															

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Halides (cont'd)	Vinylidene Chloride	75-35-4															
Hydrocarbons	Benz[*a*]anthracene	56-55-3					Y										
	Benzene	71-43-2	Y	Y	Y			38	Y	Y	Bio	N	Y				
	Benzo[*a*]pyrene	50-32-8					Y		Y		Bio	Y					
	Benzo[*b*]fluoranthene	205-99-2															
	Biphenyl	92-52-4											D			Y	100
	Butadiene, 1,3-	106-99-0	Y	Y				12	N	H		Y	Y				
	Chrysene	218-01-9							Y								
	Dibenzo(a,h)anthracene	53-70-3															
	Ethylbenzene	100-41-4		Y				31						Y	300	Y	1,000
	Fluoranthene	206-44-0							Y	L	Bio	Y					
	Hexane	110-54-3						31						Y	300		
	Indeno(1,2,3-cd)pyrene	193-39-5							Y	L	Bio	Y					
	Naphthalene	91-20-3		Y				22	Y	M	Bio	Y					
	Phenanthrene	85-01-8					Y		Y	L	Bio	N					
	Styrene	100-42-5		Y				17						Y	30	Y	1,000
	Toluene	108-88-3		Y				52	Y	H	Bio	Stable					
	Trimethylpentane, 2,2,4-	540-84-1														Y	100

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values				
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Hydrocarbons (cont'd)	Xylene, o-	95-47-6		Y				43	Y	M	Bio	N	D			Y	100
	Xylene, —	108-38-3		Y				43	Y	M	Bio	N	D			Y	100
	Xylene, p-	106-42-3		Y				43	Y	M	Bio	N	D			Y	100
	Xylenes	1330-20-7		Y				43	Y	M	Bio	N	D			Y	100
Metal/Minerals	Antimony Trioxide	1309-64-4												Y			
	Arsenic +3	7440-38-2+3						16					Y			Y	3
	Arsenic +5	7440-38-2+5	Y	Y				16					Y			Y	3
	Asbestos	1332-21-4	Y	Y													
	Attapulgite	12174-11-7															
	Beryllium Compounds	7440-41-7	Y	Y				11					Y	Y	10	Y	300
	Cadmium Compounds	7440-43-9				Y	Y	14					Y			Y	10
	Chromium +6	7440-47-3+6	Y	Y				19					Y			Y	300
	Chromium +3	7440-47-3+3	Y	Y				19								Y	100
	Erionite	12510-42-8															
	Hydrochloric Acid	7647-01-0						26	N	L		Y		Y	1,000		
	Lead	7439-92-1	Y	Y	Y		Y	19									
	Manganese Compounds		Y	Y													
	Mercuric Chloride	7487-94-7		Y			Y		N	H	Bio	Y				Y	1,000

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Metal/Minerals (cont'd)	Nickel	7440-02-0	Y	Y				19									
	Nickel Subsulfides	12035-72-2	Y	Y				19									
	Nickel Subsulfides	12035-72-2	Y	Y									Y				
	Nickel Chloride	7718-54-9	Y	Y				19								Y	300
	Nickel Sulfate	7786-81-4	Y	Y				19								Y	300
	Selenic Acid	7783-08-6															
	Selenious Acid	7783-00-8											D			Y	3
	Selenium Dioxide	7446-08-4															
	Selenium	7782-49-2											D			Y	3
	Silica	14808-60-7															
	Sodium Selenide	1313-85-5															
	Sodium Selenate	13410-01-0															
	Sodium Selenite	10102-18-8															
	Talc	14807-96-6															
Organic Acids	Acetamide	60-35-5															
	Acetonitrile	75-05-8							Y	H	Bio	N		Y	100		
	Acrylamide	79-06-1											Y			Y	1,000
	Acrylic Acid	79-10-7												Y	1,000	Y	100

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	Chemical Name	CASRN	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Organic Acids (cont'd)	Acrylonitrile	107-13-1	Y					12	M	M	Bio	Y	Y	Y	1,000		
	Bis(2-ethylhexyl)phthalate (DEHP)	117-81-7							Y	L	Bio	Y				Y	1,000
	Caprolactam (DELETED)	105-60-2														Y	100
	Captan	133-06-2														Y	100
	Dibutyl Phthalate	84-74-2											D			Y	1,000
	Dimethyl Phthalate	131-11-3															
	Dimethylcarbamoyl Chloride	79-44-7															
	Dimethylformamide	68-12-2												Y	300		
	Ethyl Carbamate	51-79-6							N	L	Bio	Y					
	Ethyl Acrylate	140-88-5															
	Maleic Anhydride	108-31-6							N	M	Bio	Y				Y	100
	Methyl Isocyanate	624-83-9							N	M	Bio	Y					
	Methyl Methacrylate	80-62-6															
	Methylenediphenyl Diisocyanate	101-68-8												Y	300		
	Toluene Diisocyanate	26471-62-5												Y	30		
	Vinyl Acetate	108-05-4							Y	H	L	Y					

TABLE B-1 (cont'd). AIR TOXICS

			Program Objectives						Physical Properties					Risk Values			
Group	**Chemical Name**	**CASRN**	Urban	Mobile Source	Indoor Source	Great Waters	PBT	MACT	Lipophilicity	Fugacity	Sorption	Chemical equilibria	Cancer Unit Risk	RfC	RfC Uncertainty	RfD	RfD Uncertainty
Phenols and Derivatives	Carbaryl	63-25-2														Y	100
	Catechol	120-80-9															
	Cresol, *m*	108-39-4							Y							Y	1,000
	Cresol, *p*	106-44-5							Y								
	Cresol, *o*	95-48-7							Y							Y	1,000
	Phenol	108-95-2						19	Y	M	Bio	Y	D			Y	100
	Propoxur (Baygon)	114-26-1														Y	100
Sulfur- and Phosphorus-Containing	Carbon Disulfide	75-15-0						12						Y	30	Y	100
	Carbonyl sulfide	463-58-1															
	Ethylenethiourea	96-45-7														Y	3,000
	Hexametapol	680-31-9															
	Phosphine	7803-51-2											D	Y	1,000	Y	100
	Propane Sultone, 1,3-	1120-71-4															

low bioavailable (L). And, chemical equilibria of the compounds indicates the potential for the HAP to speciate and transform in the atmosphere or other environmental media, as yes (Y) or no (N).

In addition, Table B-1 lists information about the risk values for the HAPs. The IRIS database was searched for the availability of cancer unit risk factors, RfCs, and RfDs. Some IRIS assessments were available but had high uncertainty factors (>1,000) and, thus, also included as HAPs needing further dose-response assessment development. Current assessment activities in ORD also were sought out to determine those cancer and noncancer dose-response assessments underway.